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COMMUNICATIONS

NATIONAL POLICY TO BE ADOPTED IN A TROPICAL COUNTRY FOR THE PREVENTION OF BLINDNESS*

BY

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LONDON

NEARLY five thousand years ago, during the IIIrd Dynasty, there lived in Egypt a physician named Imhotep, whose reputation was such that he became an administrator of the realm, and finally was deified. However, it was not until the advent of the VIth Dynasty that the earliest known oculist of human history became known. His name was Pepi-Ankh or Iri, and his funeral stele was found near the Pyramids of Giza.

The hieroglyphic script of the Ebers papyrus, which is now at Leipzig, was written about 1500 B.C. It contains directions for the treatment of ocular diseases, culled from books of the XIIth and XIIIth Dynasties; from its perusal the deduction may be made that acute ophthalmias, trachoma and blindness had been prevalent in Egypt since times which, even then, were distant.

We have the authority of Homer for the statement " In Egypt men are more skilled in medicine than any of the human kind." This occurs in the Odyssey (Book IV, line 227), and was written

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or sung on the Western Sea-board of Asia sometime before the Eleventh Century, B.C. Since I had the privilege of teaching ophthalmology in Egypt for a period of twenty years, I like to think that Homer's words are still true of modern Egyptian oculists.

During ancient times there was a great difference between the status of the medical profession in Egypt and that in countries further East. In Egypt there were able practitioners, who had the confidence of the people, while in Assyria it was necessary to threaten the surgeon with the loss of an eye or a hand if he failed in his proposed operation. In later times, Cyrus, King of Persia, requiring the services of an oculist, sent for one from Egypt, just as did His Majesty King Ibn Saud of Arabia more recently.

Since we are meeting in this ancient land of Egypt, where history may be learnt from the glorious monuments erected during epochs when Europe was inhabited by savage herdsmen, it may be permitted to refer to the later ophthalmological history of the country in so far as it impinges on the causes of blindness.

About the time that Euclid studied the first elements of optics at Alexandria, at the end of the fourth century, B.C., Herophilus wrote a book on the eyes, which unhappily has not been preserved.

In the beginning of the second century, B.C., Heliodorus, who practised at Alexandria, wrote a work on surgery, a fragment of which has survived, describing an operation for ingrowing eyelashes and various remedies for ophthalmia.

In the seventh century of the present era Paul of Aegina, who lived at Alexandria, made a compilation from works of the great Graeco-Roman doctors of earlier times for three ophthalmological chapters of his book on medicine. He described the operation for ingrowing eyelashes which was in common use in Egypt thirty or forty years ago. The operation consisted in an attempt to evert the ingrowing lashes of the upper lid by the removal of a piece of skin; this was effected by including a fold of skin between two pieces of reed or stick which were tied tightly together at their extremities. Since its blood-supply was cut off, the skin necrosed and became detached after some days. The inversion of the lid was occasionally cured, but always at the expense of a shortened upper lid, which no longer sufficed to cover the eye, blindness frequently resulting. Such cases were seen frequently thirty years ago, but their quack perpetrators were ruined by the advent of the Egyptian Ophthalmic Hospitals.

Between the VIIIth and XIVth centuries there were numerous Arab ophthalmologists who left treatises which are still extant.

Thus the medical literature of the Arabs not only adopted and preserved the ophthalmic lore of the Graeco-Roman physicians

of an earlier epoch, but considerably enriched it at a period when in Europe all the sciences were in a state of profound decadence. It should be particularly noted that both Greek and Arab surgeons distinguished exactly between chronic trachoma and acute ophthalmia, a clarity which disappeared with the decline of Arab science.

From these times onward until the commencement of the XIXth century the history of ocular science is vague throughout the world.

Much has been written about the ophthalmias which ravaged the European armies during the first half of the XIXth century. This subject has been ably described by Meyerhof, and is of importance because it was during this time that their contagious nature was first appreciated. This theory, for so it was considered at the time, was first suggested by Dr. Brigges, the surgeon of the warship Ajax, when stationed at Alexandria in February, 1802. In the same year Dr. Edmonston, a young Scottish surgeon of the second regiment of the Argyllshire Fencibles, published a communication on the contagious nature of ophthalmia.

In his " History of the Expeditionary Force in Egypt," published in 1803, Lieutenant-Colonel Wilson referred to the horrors of ophthalmia. He said: " Lately some extraordinary cases have appeared which prove that ophthalmia is highly infectious." So this cavalry officer seems to have been very receptive of new ideas, and was not afraid to counter the generally accepted medical opinion of the time.

It is chiefly in the light of our present-day knowledge of the cause of conjunctival infections in Egypt, that we are able to make a retrospective diagnosis of the eye diseases which raged in the British and French armies at that time.

In 1801 more than 3,000 French soldiers were attacked by ophthalmia without a single one losing his sight. This accords with what we know of the period of occurrence and course of conjunctivitis due to infection with the Koch-Weeks bacillus. The British troops suffered more severely, from what must have been gonococcal ophthalmia, for this is a more formidable enemy than the Koch-Weeks infection.

The subsequent history of large numbers of British and other European troops showed that after the acute ophthalmia had subsided, the chronic disease, trachoma, made its appearance. When these troops were disbanded they propagated trachoma among their families, and by these means the disease became widespread throughout Europe.

However, it must not be supposed that the Napoleonic wars introduced trachoma into Europe. Already Italy, if not other countries, had been trachomatized as the result of the return from Palestine of infected Crusaders.

Causes of Blindness in a Tropical Country

It is obviously necessary to determine the causes of blindness before enunciating any national policy to be adopted in a tropical country for its prevention. Even before this we must say what we mean by blindness. In the statistics of the Egyptian Government Ophthalmic Hospitals, since the year 1908 up to the present time, the definition adopted has been that suggested by the great French surgeon, Trousseau, namely, inability to count the fingers of a hand held up in front of the eye at a distance of one metre. In highly industrialized countries, and where the tabulation of statistical details has reached a fine art, much more elaborate definitions and data are employed. However, it is probable that in their simple form the Egyptian statistics give an exact idea of the common causes of blindness in the country. In my opinion none of the International Classifications of Blindness which have been suggested is applicable to tropical countries.

An examination of the statistics of the Ophthalmic Hospitals of Egypt for the year 1933 shows that out of 838,625 patients examined the number of those blind in one or both eyes was 60,157.

Causes of Blindness

Congenital	6
Acquired:	
Conjunctivitis	48,459
Choroid, retina and optic nerve	558
Primary glaucoma	5,529
Injury or operation	405
Cataract	2,632
Infectious diseases	27
Iritis, endogenous	1,786
Unclassified	755
	60,157

Various forms of conjunctivitis, including the sequelae of trachoma accounted for more than 80 per cent., primary glaucoma for about 9 per cent., cataract for about 4 per cent., and iritis for nearly 3 per cent.; while other causes were less than 4 per cent.

As far as is known at the present time there are no means for the prevention of the development of glaucoma or cataract, and the causes of the endogenous iritis in Egypt have not been sufficiently studied; it is therefore left to us to devise means for the prevention of the dangers attendant on inflammation of the

conjunctiva, that is to say of the mucous membrane which lines the eyelid and the globe of the eye, and shuts off the interior tissues of the orbit from the atmosphere.

Before undertaking a consideration of this subject it is of interest to compare the incidence of blindness in some European countries with that in countries further East. It is usual for statisticians to consider the rate per 100,000 of the population, and that is done here: In England and Wales it is 119, in France 73, and in Holland 55; it is to be remembered that in England and in France there are a large number of industrial accidents resulting in blindness but apart from a diminishing amount of ophthalmia neonatorum there are comparatively few purely conjunctival causes.

Now note the difference in countries where conjunctival diseases are rife: Palestine 843, Egypt 776, Cyprus 730 (all from Census Reports), and India (according to the estimate of the Director General of Medical Services, Major-General Sir John Megaw), 570.

It is clear that blindness is much more common in countries where inflammation of the conjunctiva is of frequent occurrence. Now this conjunctivitis is of two kinds, the acute variety, in which the eye may be rapidly destroyed, and the other is a special form of chronic conjunctivitis called trachoma, which is not of bacterial origin. The action of these two forms of inflammation will be briefly described.

Acute Conjunctivitis as a Cause of Blindness

Ophthalmia neonatorum or "babies' sore eyes" is very common in India among newly born infants. It is the result of infection from the maternal passages during birth. The causative organism is usually the gonococcus, but may be one of several other varieties of bacteria, while occasionally the inflammation may occur in the absence of any bacterial organism.

While ophthalmia neonatorum is very rare in Egypt and Palestine, children early fall victims to other forms of conjunctivitis, due to the pneumococcus, the gonococcus, the Koch-Weeks bacillus or the diplobacillus of Morax-Axenfeld. These may occur in acute or chronic form. All are endemic throughout Egypt, and may be found at all times of the year.

It has been well-known for centuries that acute inflammations of the eye were especially prevalent during the summer in Egypt, but the seasonal variations of the Koch-Weeks bacillus and the gonococcus have been more closely studied by Meyerhof, by myself and by Wilson.

In the coldest part of the year, January and February, there is

very little acute conjunctivitis of any kind. As the weather gets warmer the incidence of Koch-Weeks conjunctivitis increases, and reaches its maximum in May or June. After this there is a marked falling off in the number of cases seen, although the temperature remains high; but about the middle of August there is an exacerbation, but this is never so great as the earlier one.

Very few cases of gonococcal conjunctivitis are seen during the winter; indeed it is not until June that they increase; then after a temporary check they reach a maximum in October.

Wilson has offered an interesting explanation of these seasonal variations: first in the case of the Morax-Axenfeld bacillus, the increase of inflammation due to this organism coincides with two factors, the increase of the atmospheric temperature, and the first fly-breeding season. Then with increased dessication due to heat there is a diminution both of flies and of ophthalmia, until the rise of the Nile produces greater humidity with the second fly-breeding season beginning about the middle of September, when there is another exacerbation. This is followed by a gradual fall until the month of December.

Second in the case of the gonococcus, the incidence remains low until there is a certain degree of heat, as in July. Then there is some diminution in the cases due to this organism, owing to the fact that it does not stand drying very well, nor do the flies. Later when there is much greater humidity, and the second fly-breeding season having begun in October, there is a vast increase of gonococcal ophthalmia. When the temperature falls at the end of October and beginning of November there is a very rapid diminution. However, there are always a certain number of cases which last throughout the winter, and form a reservoir of infection.

It is thus seen that the seasonal epidemics are closely related to the three factors: temperature, humidity and flies.

Trachoma as a Cause of Blindness

Uncomplicated trachoma is typically a quiet inflammation of the mucous membrane of the eyelids which spreads quickly to the cornea, causing a vascular opacity called pannus. It is transmitted mainly by the fingers and by such articles as towels; it is probable that flies may convey the contagious material from an infected to a healthy eye, but this is not the rule. Trachoma is not due to the action of bacteria but to that of a virus, as is the case with small-pox and with the foot and mouth disease of cattle.

It is probable that trachoma arose among the nomad races of the plains of Mongolia. From here it spread westward with the Mongol invasions, infecting the inhabitants of every country

through which they passed, until it reached the Mediterranean Sea, around the shores of which it reached its apogee.

Travelling eastward through China and Siberia with the vast migrations which crossed to the American continent, the disease found another permanent home among their descendants, the American Indians.

Such a theory of the dispersion of trachoma, explains its world wide distribution.

Uncomplicated trachoma may be the cause of very serious depreciation of visual acuity, though improvement may occur after treatment. However, when the disease has spread to the supporting structure of the eyelids, as usually happens after the disease has been present for some time, they become thickened and their lashbearing margins become inverted. The eyelashes then play upon the surface of the eye and abrade the cornea. While such an abrasion is merely superficial, and if the lid inversion is corrected by operation, it may be healed readily without loss of transparency of the cornea. If, however, the inversion persists, and especially if the abrasion, now having become an ulcer, becomes infected with dangerous bacteria, complete destruction of the eye may result. Indeed this is what happens in 80 per cent. of the cases in Egypt and Palestine.

The virulent effects of trachoma in a heavily trachomatised country are exhibited with great clarity by Wilson as the result of his examination of the inhabitants of a village, Bahtim, within some miles of Cairo. This village had 3,549 inhabitants, of whom 491 were absent and not examined; also 140 babies under one year old were excluded, and formed the subject of a separate study. It was found that 97·6 per cent. had trachoma, and 2·4 per cent. had acute conjunctivitis. Of the trachomatous subjects who had cicatricial changes in the conjunctiva 21 per cent. had ingrowing eyelashes, and 4 per cent. had been operated on already for this sequela. I have already referred to the danger to the eye of ingrowing eyelashes, so it is not surprising to learn that 5·5 per cent. were blind in one eye, and 1 per cent. blind in both eyes.

There is no reason to suppose that ocular conditions in other villages of Egypt, and in other countries where trachoma and acute ophthalmias coexist, differ very much from those of Bahtim. (See " Trachoma " by A. F. MacCallan. Butterworth and Co., 1936.)

Other Causes of Blindness in Tropical Countries

Small-pox used to exact a heavy toll of blindness in Egypt and in Palestine, but since the introduction of compulsory vaccination this has disappeared. In India and other oriental countries,

where vaccination is not universal, small-pox is still a fruitful cause of blindness.

The industrial causes of blindness, that is accidents in factories and workshops, are much less frequent in Egypt and India than in more highly industrialised countries, such as England, France and Germany, although with increasing industrialisation there is no doubt that they will increase.

In India a starvation disease called keratomalacia is a very common cause of blindness among young children in some parts. The conjunctiva becomes dry and greasy-looking, the cornea quickly undergoes ulceration, and blindness frequently results. It is due to a lack of the vitamins contained in milk, butter, and fresh vegetables. In the case of breast-fed babies the condition may be cured, if taken early enough, by giving the mother cod-liver oil.

Squint is a more frequent cause of blindness in the East than it is in European countries owing to the frequency of corneal opacity, which prevents the co-ordination of the two eyes.

Among conditions which cause blindness in tropical countries there are others which cannot be considered now, such as retinitis pigmentosa due to deficiency of vitamins in the diet, pyorrhoea which is a stimulus to iritis, and leprosy.

N.B.—*It is particularly to be noted that blindness is much more common among a rural population than among town-dwellers.*

The Means of Prevention

The means of prevention embrace many medical, sanitary and social measures, the value of which differs according to the geographical position of a country and the cultural standards of its inhabitants; also the causes of blindness differ in different countries.

I shall describe the means of prevention which exist in Egypt, and for which I have been largely responsible.

(1) *Hospitals.*—In 1903 Sir Ernest Cassel, who had financed the building of the Asswan Dam, created a trust fund of £40,000 " for the purpose of training Egyptian doctors in the special work of ophthalmology, in order that their poorer fellow-countrymen might be able to receive from them gratuitous advice and treatment for the all-pervading ocular diseases."

It was decided to create a travelling ophthalmic hospital under canvas where young Egyptian doctors might practise under the direction of a British Ophthalmic Surgeon, and I was invited to inaugurate the work. The success of the hospital was immediate, and its popularity was so great that it was frequently impossible to treat all cases; when this became known men suffering from

ingrowing eyelashes sometimes fought to get the next turn in the operating tent.

A second hospital was added in the following year, and in 1906, as the result of my advocacy, money was granted to build, equip and maintain two hospitals, one in Lower and one in Upper Egypt, and they all became part of the Egyptian Department of Public Health.

Later when credits for new hospitals could not be granted, I obtained an undertaking from the Government that credits for maintenance should become available for hospitals built by local effort in each of the fourteen provinces of Egypt. These hospitals were all completed before I left Egypt in 1924. The capital sums required were raised through my influence by subscription, or from the funds of the Provincial Councils.

The need for further means of ophthalmic relief in the densely populated country districts enabled me to organise several more permanent and travelling hospitals, entirely paid for out of local funds.

All the hospitals are staffed by Egyptian oculists, who engage in the practice of no other branch of medicine.

During his latter years the influence of His Majesty King Fuad the First gave powerful assistance to the campaign against blindness in Egypt.

The Ophthalmic Section of the Ministry of Health administers the hospitals, of which 25 have been specially designed and built, 15 are travelling hospitals under canvas, and 38 are ophthalmic departments of General Hospitals.

An immense amount of ophthalmic work of all kinds is carried out at these hospitals, where in 1934 approximately a million patients were examined, and 100,000 operations performed.

There has been no change in hospital policy since I resigned from the service of the Egyptian Government in 1923, and the present administration of the hospitals gives me great satisfaction.

It is especially gratifying to me that the system of hospital and school statistics which was introduced in my first printed Report in 1911 is still being used, and I have quoted from them several times in this paper. I wish to make my acknowledgments for this to the Under Secretary of State and the Director of the Ophthalmic Section of the Ministry of Public Health.

I have always insisted on the importance of establishing a hospital under the charge of an ophthalmic surgeon, as a preliminary to any campaign for the reduction of blindness and eye-diseases, wherever such is not already in existence. Its place can be taken, though less satisfactorily, by the ophthalmic department of a general hospital. It is to be understood that I am speaking about Eastern lands, where acute conjunctival affections

are so prevalent that patients suffering from them largely outnumber patients suffering from general diseases.

The ophthalmic hospital must be in the clinical charge of an ophthalmic surgeon who is prepared to undertake the diagnosis and surgical treatment of all diseases of the eye. My reason being that if the father of a family has ingrowing eyelashes causing pain and the gradual onset of blindness, and is unable to obtain relief at a hospital where a surgical operation can almost instantly cure him, he is not likely to pay much attention to the ocular disability of his little daughter who has a slight discharge from her eyes, caused by conjunctivitis, which at any moment may become acute and lead to ulceration and blindness.

It is to be noted that in the terms of Sir Ernest Cassel's original gift all treatment in the hospitals is entirely gratuitous, and is provided for the poor. Such persons who are in a position to pay for treatment are directed to the private clinic of the ophthalmic surgeon, which is not in connection with the hospital enclosure.

(2) *Pathological and bacteriological work.*—During the earlier years of the history of the hospitals the examination of pathological and bacteriological materials was carried out in small laboratories, one in Upper Egypt and one in Lower Egypt. Later a centralised laboratory and library were established at one of the hospitals near Cairo, which was built and equipped at the expense of my personal friends and of the ophthalmic surgeons who worked under my direction. This did excellent work until the opportunity presented itself of establishing a first-class laboratory where research work could be carried on in a more elaborate manner than had been possible before.

(3) *Memorial Ophthalmic Laboratory.*—The Imperial War Graves Commission decided to erect some Memorial to the men of the Egyptian Labour Corps and Egyptian Camel Transport Corps who gave their lives during the Great War, while serving with the Armies of the British Empire. I placed before the Commission the project of an ophthalmic laboratory which would serve the clinical and scientific needs of the Egyptian Ophthalmic Hospitals. Owing to the support given to my project by His Excellency the Prime Minister, a sum of £6,600 was granted. I then obtained a grant of land at Giza on the outskirts of Cairo from the State Domains Administration on which to build the Laboratory. This was situated adjacent to the new King Fuad the First Ophthalmic Hospital at Giza, which I had designed. I drew up the interior plans of the laboratory, which the architect incorporated in magnificent memorial form. I asked for and obtained a grant of £2,000 from the Ministry of Finance to equip the laboratory adequately with all the most recent instruments,

and arranged for the income of the Cassel Fund to be used for the maintenance of the laboratory and for the salaries of the staff; to this was afterwards added the interest of a trust fund established by the British Red Cross, and a grant from the Egyptian Government. His Excellency the British Ambassador is the trustee of the two funds and the administration is in the hands of the Ministry of Health of the Egyptian Government.

The success which attended the work of the laboratory is due to the appointment as Director of Dr. Wilson who publishes an annual report containing an account of the clinical researches on which he has been engaged, from which I have quoted several times in this report. In this he has the assistance of the pathologist, Major Stewart, formerly of the Indian Medical Service.

Besides the research and pathological sections of the work, the laboratory is the centre for post-graduate ophthalmic education, for which there are two courses every year. Between 15 and 20 doctors attend each course.

(4) *Prophylaxis of blindness in day schools.*—In countries where inflammatory affections of the conjunctiva are prevalent the inception of some form of regular treatment in day schools is of the greatest educative value. It teaches the pupil to become eye-conscious, and it is not only they who learn, but also the inhabitants of the home of each pupil.

There is a National Egyptian Committee for the Prevention of Blindness. The official Public Health organisation and the Egyptian Society for the Welfare of the Blind take a particular interest in the study of this question.

In 1907 I obtained permission to carry out regular ophthalmic inspection at a school of 464 boys, every one of whom I personally examined by eversion of the eyelids at the beginning and ending of each school session. The operative and medical treatment was carried out by an ophthalmologist on five days a week. The experience was of vital interest to both inspecting surgeon and to the local ophthalmologist. The value of the arrangement was soon recognised by the Educational authorities, and now 32 schools are inspected and treated. During the year 1934-1935 10,459 boys underwent inspection and treatment.

Although 98·67 per cent. were trachomatous, it was found that 95·18 per cent. had both corneae clear to the naked eye on focal illumination at the end of the school year, a satisfactory result of the year's work.

It may be noted that in spite of the ophthalmic campaign, which I inaugurated more than thirty years ago, practically all the boys in the Government Primary Schools show evidence of trachomatous infection. This universal incidence includes boys

who merely exhibit slight stigmata of healed trachoma (Trachoma stage IV).

It is correct to class as trachomatous every individual who exhibits any sign of active or healed trachoma, for in many cases it is impossible to state that an exacerbation or a fresh infection will not occur.

Since infection with trachoma occurs in the family, about 25 per cent. of children becoming infected before the age of one year, it appears to be a permanent scourge inflicted on the country, and the only thing that can be done by ophthalmologists and hospitals, is to minimise the ill effects of the disease, and prevent the occurrence of complications. The importance of sanitary measures in the prophylaxis of trachoma and in the prevention of the blindness due to it, will be discussed later.

(5) *Children's Dispensaries and Ophthalmic Welfare Clinics.*—Dispensaries for children, under the charge of qualified British nursing sisters were introduced in several towns in Egypt by the late Lady Cromer. They have been of the greatest value, in the treatment of general and eye diseases and in giving advice to mothers as to children's diet.

During a period of reorganisation I had the pleasure of giving some help to the Committee of Management.

Two of these Children's Dispensaries, one in Cairo (Madbouli) and the other on the road to the Pyramids (El Haram), are managed by a Committee of British ladies, and are staffed by British Nursing Sisters.

The Children's Dispensaries in the Provinces are administered by the Provincial Councils under the direction of the Mudir or Governor of the Province. They are managed by Egyptian Nursing Sisters (Hakimas).

(6) Other hospitals where eye treatment is carried out in Egypt are numerous, such as the Cairo Hospital of Kasr-el-Aini, the ancient Qalaoon Hospital also in Cairo, the Church Missionary Society's Hospital in Old Cairo, the Municipal Hospital of Alexandria, and some others.

(7) There are many classes of social work which can be carried on by non-medical organisations which are calculated to aid in the prevention of blindness in tropical countries.

Such work is carried on by the Indian Red Cross and by its Junior branch. Great credit is due to those who give their time or money to the work of these or analogous organisations in other countries.

The International Association for the Prevention of Blindness is of great importance, but I can only refer to it briefly. The mainspring of the Association for eight years has been Professor de

Lapersonne, and now Dr. Bailliart is giving his time, and using his undoubted genius for the same object. The Association is extremely fortunate in having the services of Dr. A. Churchill as Associate Secretary General; it is difficult to imagine a more efficient or more agreeable officer.

La Ligue contre le Trachome was founded by Morax and Nicolle, whom we have no longer with us. Again Dr. Bailliart has undertaken the care of La Ligue, with the help of Dr. Paul J. Petit. Its publication, *La Revue Internationale du Trachome* has also become the organ of the *International Organisation against Trachoma,* and is exerting a great influence throughout the ophthalmic world in reducing the loss of visual acuity due to trachoma.

There are other branches of social work which are of great value which will be dealt with by other reporters, but with which I have no personal experience.

(8) *Treatment in remote districts by non-medical assistants.*—The value of children's dispensaries conducted by qualified nursing sisters has already been noted; some allusion must be made to the allocation of native-trained hospital attendants to remote country districts. The idea is that such men should be supplied with simple lotions and eye drops, and receive some instruction in first aid treatment for acute ophthalmias and for cases of trachoma. I can imagine that they would be of some service if they were kept under rigorous discipline by the frequent inspections of an ophthalmologist, and provided that they had not a number of extraneous duties to perform. The difficulty is that such men can with difficulty be prevented from claiming backsheesh for their services, and if they meet with any success they set up as eye-doctors, if not as physicians.

(9) *Sanitary measures.*—By the application of modern sanitary measures, such as are known in rich and highly civilised communities it would be possible to reduce the incidence of blindness in tropical countries to a tenth of the existing figure. Such measures would include good dwelling accommodation, a water-supply and the provision of latrines and drainage. Imagine how possible it is to envisage these at some village in Southern Palestine, at El Deir opposite the Temple of Abu Simbel, at El Badari opposite Assiut, or at some village in the plains of Central India!

In places where ophthalmias are now prevalent: where water is scarce, where dwellings are mere mud huts; and where neither latrines nor any system of drainage has ever been heard of, the suggestion of making sanitary improvements at the present day sounds fantastic.

Yet without such sanitary improvements no amount of medical aid, whether by hospitals or dispensaries, will make any marked impression on the incidence of blindness, nor on the liability of the children to contract trachoma at an early age.

Conclusion

The value of the ophthalmic campaign in Egypt may be judged by the percentage of persons, blind in one or both eyes, who present themselves at the hospitals and crave treatment.

Yearly percentage of blindness among the patients of the Egyptian Government Ophthalmic Hospitals

YEAR	Percentage of blindness in one or both eyes including cataract cases
1911	19·2
1912	15·2
1913	14·8
1914	13·2
1915	12
1916	11·2
1917	13·9
1918	14·6
1919	15·3
1920	13·6
1921	12·2
1922	11·8
1923	11·2
1924	10·8
1925	10·4
1926	10·1
1927	9·8
1928	9·3
1929	8·4
1930	8·4
1931	7·3
1932	6·9
1933	6·7
1934	6·2

In the year 1911 this percentage was 19·2; it has been gradually reduced, until in 1935 it was 6. So that in less than a quarter of a century the percentage of blindness among hospital patients has been reduced by nearly one third.

I therefore think that the system adopted for the Egyptian Ophthalmic Campaign has been justified by results.

"THE NATURE OF THE AQUEOUS HUMOUR"*

BY

J. DOUGLAS ROBERTSON

LONDON

IN a recent issue of this Journal (Vol. XXI, p. 577), Duke-Elder has raised several points in criticising my paper concerning the theories on the formation and exit of the aqueous humour.

1. *The canal of Schlemm.*—Duke-Elder has written (*Brit. Jl. Ophthal.*, Vol. XXI, p. 578)—"the essential feature which enables this structure to function as a preferential drain for fluids in the eye is that while it (like the capillaries) has an endothelial wall through which fluid traffic can take place, it . . . must have a pressure considerably less than the capillaries. It is thus a capillary (in the physiological not the anatomical sense) in the venous system."

Under normal pressure conditions within the eye, it is generally believed that the intra-ocular pressure exceeds the pressure in the canal of Schlemm, but as no-one has ever measured the capillary pressure nor that in the canal of Schlemm, it has not been proved as Duke-Elder asserts that the pressure in the canal of Schlemm is *considerably less* than the capillary pressure. Indeed, it is probable that the pressures within these vessels may not be static. For in the case of the capillaries, the work of Rous and his associates suggests that the attractive conception of a circulation of fluid through the tissues with filtration at the arterial end of the capillary and absorption at the venous end may not be true. They believe that the capillaries throughout their entire length are either producing filtrate or absorbing fluid, so that the hydrostatic capillary pressure at one time exceeds the colloid osmotic pressure of the plasma and at another time is less than it. That the interchange of fluid within the eye is not governed by such a law is apparent when an increase in intra-ocular pressure takes place if only the exit at the canal of Schlemm is blocked (the canal of Schlemm only forms a small part of the whole surface of the eye).

If the pressure in the canal of Schlemm exceeds the intra-ocular pressure, why does fluid not enter the chamber from the canal? There is no evidence that it does so. It may be argued that by reason of the alleged protein rich fluid within the canal of Schlemm an osmotic force comes into play. But the fact remains as I have pointed out (*Brit. Jl. Ophthal.*, Vol. XXI, p. 444), if the canal of Schlemm could attract fluid from the anterior

* From the Courtauld Institute of Biochemistry, Middlesex Hospital, London, W.1

chamber by osmosis, it must do so by reason of its protein content, and this in the capillary is provided for by the plasma. Normally, no red cells have been observed in the canal of Schlemm and this strongly suggests that the canal of Schlemm does not conform to the requirements of a capillary and thus cannot play any part as such in the interchange of fluid by osmosis. A vessel which (*a*) permits fluid traffic in one direction only, (*b*) does not normally contain red cells, and (*c*) begins as a cul-de-sac (*i.e.*, there is no afferent vessel) and enters the venous system, is not a vascular capillary in any sense physiological or anatomical as Duke-Elder has written. Venous capillaries, or more correctly, sinusoid vessels (examples of which are found in the liver) have a relationship to the vascular system in no way different from that of the other capillaries throughout the body except that in this case the afferent vessel of the capillary is a vein and not an artery.

2. *The chemical equilibrium.*—The analyses on sodium and chloride which have been carried out by many workers and most recently by Duke-Elder and his colleagues, are such that the equilibrium between aqueous humour and blood may be explained by the Donnan theory. But it is agreed at the same time that the distribution of other electrolytes and non-electrolytes such as phosphate, sulphate, uric-acid, urea and sugar are not in accord with the Donnan theory. Of the many constituents analysed by Walker which I quoted, Duke-Elder has selected urea for closer study. He points out that the method employed by Walker was not so accurate as that described by Ranganathan and Sastri (*Biochem. Jl.* Vol. XXX, p. 2,135) and employed by Benham (*Biochem. Jl.*. Vol. XXXI, p. 1,157) in a study of the aqueous and serum from the same animal. Although the conductometric method would appear to be more accurate, it is not so accurate as Duke-Elder suggests. In the original paper of Ranganathan and Sastri this method is stated to be accurate to 1 per cent. using 0·1 ml. of blood as compared with the aeration titration method stated to be accurate to 1 per cent. using 3 ml. blood, but divergencies up to 4 per cent. are recorded for the above methods applied blood and urine using larger quantities than stated above. Duke-Elder then makes the following statement. " The passage of four years has changed a variation of 49 to 95 with an average of 68 per cent. (Walker's results in *Jl. Biol. Chem.*, Vol. CI, p. 269) to a variation of 80·5 to 95·7 with an average of 90 per cent. (Benham's results in *Biochem. Jl.*, Vol. XXXI, p. 1,157). What will the next four years bring? I am far from claiming finality from Benham's method." Prophetic utterances are, I think, out of place in a scientific publication but one feels tempted to reply that by excluding the analysis of Walker and others and confining the study of the urea equilibrium solely to the analyses carried out

by Duke-Elder and his collaborators one finds in 1927 (*Biochem. Jl.*, Vol. XXI, p. 66), the urea of the aqueous and blood agree to within 3 per cent., and in 1936 (*Biochem. Jl.*, Vol. XXXI, p. 1,157), the average of seven analyses of the aqueous and the blood agreed to 10 per cent. Thus it would appear that the passage of nine years has caused a disparity of 3 per cent. to become one of 10 per cent. Therefore, the passage of the next four or nine years may well prove interesting.

The main facts of scientific interest, however, borne out by Benham's results are that they confirm Walker's findings and refute those of Duke-Elder (*Biochem. Jl.*, Vol. XXI, p. 66, and *Brit. Jl. Ophthal.*, 1927, Monograph Supp. II). Both Walker and Benham found that the concentration of urea in the aqueous humour was less than that in the blood and as urea is freely dialysable *in vivo* and *in vitro* it follows that the aqueous humour is not produced by the process of dialysis. If the aqueous humour was a dialysate, the ratio of $\frac{\text{urea of aqueous}}{\text{urea of plasma}}$ would be 1, but Benham found the average of his seven cases to have a ratio of 0·9 and what is of greater importance, the range of his results was less than 1—(0·805 to 0·957).

Duke-Elder found that sugar was partitioned in approximately equal amounts (*Biochem. Jl.*, Vol. XXI, p. 66). The question of the concentration of sugar in the aqueous has now reached a very interesting phase in view of recent work upon the high concentration of ascorbic acid in the aqueous. This substance is a very strong reducing agent and Müller (*Nature*, Vol. CXXXII, p. 280), has found its concentration in the aqueous is possibly as high as 18 mgs. per 100 ml., while less than 1 mg. per 100 ml. is present in blood serum. My colleague Mr. J. Barrett (unpublished results) has found by employing the Hagedorn-Jensen or Shaffer-Hartman method that a concentration of 18 mgs. per 100 ml. of ascorbic acid had a reducing power as glucose of almost 13·5 mg. per 100 ml. Thus when a correction is made for the presence of ascorbic acid in the aqueous, it becomes necessary to state that less sugar is present in aqueous than blood.

3. *The osmotic equilibrium.*—Duke-Elder's most recent method of determining the osmotic pressure of the aqueous humour (*Jl. Physiol.*, Vol. LXXXIX, p. 61) merely corroborates his previous results. The point I made in my paper which Duke-Elder says he is unable to follow is more simply this. The aqueous humour is isotonic with blood, and so also is lymph, cerebrospinal fluid, gastric juice, pancreatic juice and hepatic bile. Osmotic pressure observations therefore, by any method, are of no value in determining whether a fluid is a secretion and not a dialysate or vice versa.

4. *The experimental work on the maintenance of the intra-ocular pressure.*—There is no need for me to repeat the results of my investigations into Duke-Elder's experimental work. Suffice it to say that in most of the experiments he recorded including what he considered a control (the injection of normal saline intravenously in an intact animal) the level of the intra-ocular pressure was not at any point in the experiment in accord with the theory of dialysis although he felt satisfied, at that time, that the correct answer had been obtained. Even his explanation (*Brit. Jl. Ophthal.*, Vol. XXI, p. 583), that " such analyses as they will bear, however, brings out one point of extreme importance, that, in comparison with tissue fluids, there is a definite time lag in the reaction of the eye," is not sufficient, for in his control experiment (*Brit. Jl. Ophthal.*, Vol X, p. 9, fig. 1), where a time lag was necessary the intra-ocular pressure did not alter. From Duke-Elder's own experiments along with my own observations on the intra-ocular pressure in oedematous nephritis, it may be said that a physical equilibrium does not exist between blood and aqueous, and the equilibrium level of the intra-ocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in osmotic pressure between aqueous and blood.

Conclusions

1. The canal of Schlemm is not a vascular capillary in any sense physiological or anatomical.
2. A further study of the urea and sugar equilibrium shows that the aqueous humour cannot be formed by dialysis because:—
   a. The concentration of urea in the aqueous is lower than that in the blood.
   b. The concentration of sugar in the aqueous is lower than that in the blood.
3. As secretions and dialysates are both isotonic with blood osmotic pressure observations are of no value in differentiating the one from the other.
4. A physical equilibrium does not exist between blood and aqueous, and the equilibrium level of the intra-ocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in osmotic pressure between blood and aqueous.

This is a further argument against the theory of dialysis.

Summary

In summary it may be stated that the observations which I reported originally refute the suggestion that the aqueous humour is a dialysate and Duke-Elder's most recent contribution does not cause me to alter this view.

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY. PRELIMINARY SURVEY OF HEREDITARY EYE DISEASES IN TASMANIA*

BY

J. BRUCE HAMILTON

HOBART, TASMANIA

(Continued)

It is not within the scope of this paper to attempt to expound the problems of Mendelian characters in regard to ophthalmology, but rather to decide whether or not hereditary eye disease in Tasmania conforms to these established laws. On the whole I must admit that they do, and this is only to be expected in view of the fact that most of the original members of these pedigrees were born within the British Isles.

Since 1900, Mendel's laws have become widely appreciated, so that patients affected with hereditary diseases are now constantly seeking advice from their medical practitioners. Thus it behoves all medical men, and especially ophthalmologists to be conversant with the various types of transmission, and I therefore propose to give a brief survey of the behaviour of Mendelian and sex-linked characteristics, for the guidance of those who may not be acquainted with these facts.

Dominant Transmission

(1) If both parents are affected—

(*a*) DD x D(R). All children affected.
(*b*) DD x DD. All children affected.
(*c*) D(R) x D(R). 3/4 children affected, 1/4 normal.

(2) If one parent is affected—

(*a*) DD x RR. All children affected.
(*b*) D(R) x RR. 1/2 children affected, 1/2 normal.

Recessive Transmission

(1) If both parents are affected—

RR x RR. All children are affected.

(2) If one parent is affected—

(*a*) DD x RR. All children normal.
(*b*) D(R) x RR. 1/2 children affected, 1/2 normal.

* Continued from p. 43.

Sex Linkage Transmission

The disease may be dominant, or recessive (generally recessive).

A. If the disease is recessive—

1. The males are affected more frequently than the females.

2. Females are of three kinds.

   (*a*) Completely normal and not a carrier. XX.
   (*b*) Completely normal but a carrier. XX.
   (*c*) Affected. XX.

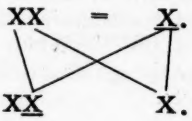
3. Males of two kinds.

   (*a*) Completely normal. X.
   (*b*) Affected. X

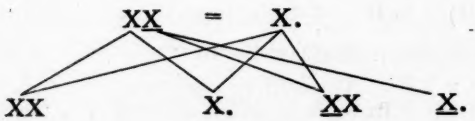
Note.—Males cannot be carriers in the sense of (2*b*).

Examples of recessive sex linkage.

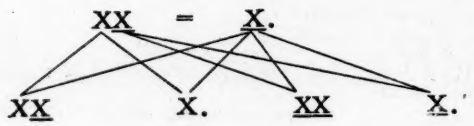
1. A female of class (2*a*) married to an affected male (3*b*) will produce normal children. All the daughters will be carriers (2*b*), and will transmit the disease to half their sons.



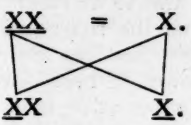
2. A carrier female (2*b*), married to a normal male will transmit the disease to half the sons. Half the daughters will be carriers (2*b*).



3. A carrier female (2*b*) married to an affected male (3*b*) will have half the daughters affected (2*c*), and the other half carriers (2*b*). Half the sons will be affected (3*b*), and half will be normal.



4. An affected female (2*c*) married to a normal male (3*a*) will hand on the disease to all the sons (3*b*), and all the daughters will be carriers (2*b*), and will hand on the disease to half the sons.



B. If the disease is dominant.

Females.

(*a*) Diseased. XX.
(*b*) Diseased. XX
(*c*) Normal. XX.

Males.

(*a*) Diseased. X.
(*b*) Normal. X.

Later when I comment on each individual pedigree which I am reporting, I shall endeavour to define the mode of transmission, but as this is only a preliminary report, and in consequence many of the pedigrees short, this will not always be possible. With regard to the brevity of some pedigrees, I am encouraged in presenting them by the fact that the Bureau of Human Heredity (115, Gower Street, London), appears to be as interested in shorter, as in longer ones, and appeals for unselected family trees (*Brit. Med. Jl.*, 1936). My fifty-three pedigrees are quite unselected.

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Astigmatism

Unfortunately in order to keep the diseases in alphabetical line I must commence with the least satisfactory of the hereditary diseases. Nor am I helped by the literature, which is extraordinarily scanty.

Astigmatism, according to Duke Elder (1934) and Ruggles Gates (1929) is undoubtedly hereditary in many pedigrees, and

is usually dominant in transmission. They state that not only is the degree of astigmatism maintained, but also the axis in most pedigrees. I have not been able to confirm the former assertion, but in pedigree 21, 8 of the 13 members of a myopic family have their astigmatism against the rule; and in pedigrees 20 and 25 every examined member has his astigmatism with the rule. But this cannot be followed in every case. The mode of transmission also is certainly recessive in some strains (pedigrees 3 and 5), but in others more data are needed to form an opinion.

With astigmatism manifesting itself in at least 3/4 of ophthalmic cases, the surgeon ultimately regards it as a necessary evil and gives it scant consideration. Nevertheless I feel if ophthalmologists working in confined areas (as I do) would pay some attention to the hereditary aspect and report their findings, that they would be agreeably surprised to find that astigmatism is not as boring as they had thought. I certainly intend to follow this aspect of heredity more closely.

As I have found some difficulty in separating the astigmatic pedigrees from those of myopia, I would suggest that these two diseases be read in conjunction rather than treating them as two separate entities as I have done. On the other hand as each characteristic has a separate gene it might have been expedient to have had separate pedigrees for each characteristic.

Comments on Pedigrees

My interest in hereditary astigmatism arose through pedigrees 1 and 2.

In *Pedigree No.* 1, Case No. I/1 was referred to me for compensation purposes. He had high mixed astigmatism with the rule, and with correction saw only 6/24 in the better eye. He attributed his defective sight to the accident, and I to inherited astigmatism. The dispute was still unsettled when his son II/2 reported with defective sight due to high mixed astigmatism with the rule, of a very similar nature to the father's. The dispute accordingly ceased.

In *Pedigree No.* 2, cases III/2 and 3, who were sisters, had high mixed astigmatism of almost identical type and strength in the four eyes. A year later the mother (II/13) called complaining of bad headaches which physicians had failed to cure. She had high astigmatism in both eyes, but especially the right. Her right refraction was simple oblique myopia, and her left simple oblique hypermetropic astigmatism. I asked her why, in view of her daughter's defect, she had not attributed her headaches to defective sight. She admitted that she had no idea defective sight was hereditary. I then made further searches and during the past

two years have collected three more pedigrees as well as those recorded under myopia.

Pedigree No. 3.—As only II/2 has been examined, it is difficult to comment with authenticity, but II/2, who is a man of wide knowledge and experience, assures me that his mother and elder sister are similarly affected. In that case the disease is most likely recessive in transmission, and I advised him to watch the eyes of his two sons.

Pedigree No. 4.—This pedigree, in part, will be discussed under nystagmus. II/3, has compound myopic astigmatism with the rule, and her left eye is amblyopic with 45° of divergence.

II/4. Has complicated cataracts from iridocyclitis.

III/8. Has mixed astigmatism against the rule.

III/60. Has left divergent concomitant strabismus, with partial amblyopia. She is emmetropic.

III/63. Has left divergent concomitant strabismus with complete amblyopia, and high oblique compound myopic astigmatism.

IV/3. Has high oblique astigmatism.

III/60 and 63 are interesting because the paternal parent belonged to the astigmatic portion of the pedigree, and the mother to the nystagmus portion. The result was squint with amblyopia —such amblyopia occuring in two distant relatives, II/3 and IV/22 (one on each branch of the family), without nystagmus, and in four other cousins (on the maternal side) with nystagmus. As members of this pedigree are constantly reporting, I may later have more definite comments to offer.

Pedigree No. 5.—This is of special interest as a member of the first family, in which at least four members have compound myopic astigmatism, married a member of the second family, of which at least three members have compound hypermetropic astigmatism. The result has been five sibs—three of which are affected with refractive errors, one with myopia in the right eye, and compound hypermetropic astigmatism in the left.

Another with pure hypermetropia, and the third with gross mixed astigmatism.

I/2. High compound myopic astigmatism with the rule and fundus changes.

I/3. Low compound myopic astigmatism with the rule.

II/3. Myopic astigmatism—no details.

II/6. Mixed astigmatism against the rule.

II/9. High compound hypermetropic astigmatism against the rule.

II/11. Low compound hypermetropic astigmatism—oblique.

II/15. High compound hypermetropic astigmatism against the rule.

III/2. Right eye, low myopia. Left eye, low hypermetropic astigmatism against the rule.

III/3. Moderate hypermetropia.

III/5. High mixed astigmatism.

The disease definitely appears to be recessive, but more than one gene is certainly involved.

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Congenital Cataract

According to Macklin (1927) it is estimated in Canada that 13 per cent. of the pupils in institutions for the blind are suffering from hereditary cataract. The proportion in Tasmania is much higher than this; 3 of the 6 blind pupils in the Tasmanian Institution for the Blind are suffering from cataract. On the other hand, Best (1934) considers that 13·7 per cent. of blindness in U.S.A. is due to cataract, but in Tasmania I find 20·45 per cent. of the blind of whom reliable clinical information is obtainable suffering from this disease. This figure appears to me to be astounding especially in view of the fact that of all hereditary eye diseases causing blindness, cataract should be the most responsive to treatment. I can account for this high percentage in Tasmania in the following manner:—

(1) Late operative interference. This is exemplified in pedigree 7, where one father (IV/14) refused to allow his children to be operated on until they had developed nystagmus and strabismus with amblyopia. In other cases operation has not been considered, due to ignorance or isolation of the parents.

(2) Incomplete operative interference. This is exemplified by pedigree 6 where III/5 had a right needling done many years ago, but did not report for glasses, which eventually gave him 6/12 partly and J. 4. Fortunately he has submitted his daughter aged 1½ years for operation, and the visual result should be good.

(3) Conservative Medical Advice. The unsatisfactory results that have sometimes followed discission of the crystalline lens has often deterred ophthalmologists from advising operative interference.

(4) The Commonwealth Government does not insist on applicants for blind pensions undergoing expert examination and treatment before being accepted. It is left to the applicant's choice between blindness with pension, and sight without pension, and unfortunately, many choose the former course. This undoubtedly should be remedied at once.

Sex Incidence

In my four pedigrees (Nos. 6, 7, 8 and 9), there are 13 affected males and 18 affected females, giving a percentage of 41·9 and 58·1 respectively. In pedigree 7, there are five members whose sex has not definitely been determined. Taking the pedigrees reported by Bishop Harman (1909), Macklin (1927), Galloway (1930), Khan (1926), Knapp (1926) and Dunforth (1914), I have found the sex incidence as 55 males and 63 females, giving a percentage of 46·6 and 53·4 respectively, which conforms with the predominance of females shown above.

Age of Onset

Loy (1936) quotes Nettleship's six laws pertaining to the occurrence of cataracts in females, and the 5th law states that—" the age of onset is approximately the same in persons of the same generation." In my three pedigrees, so far as I am able to ascertain, every case was apparent from infancy, with the exception of III/23 of pedigree 8, where the opacities are only partially developed at puberty.

Type of Opacity

According to Clapp (1934) congenital cataracts may take 10 partial forms, and of course one complete form, but nowhere in his book can be found a discussion on the hereditary nature of these opacities.

My longest pedigree, No. 7, exhibited zonular opacities almost uniformly with occasional dislocation of the lens, and in one case at least the lens had been practically absorbed. In pedigree No. 6 the two cases examined were of coralliform type, while pedigree 8 exhibited no uniformity in the two cases.

I have used the word " zonular " in reference to pedigree 7 intentionally, as I feel the word " lamellar " should be left for those cataracts following infantile tetany.

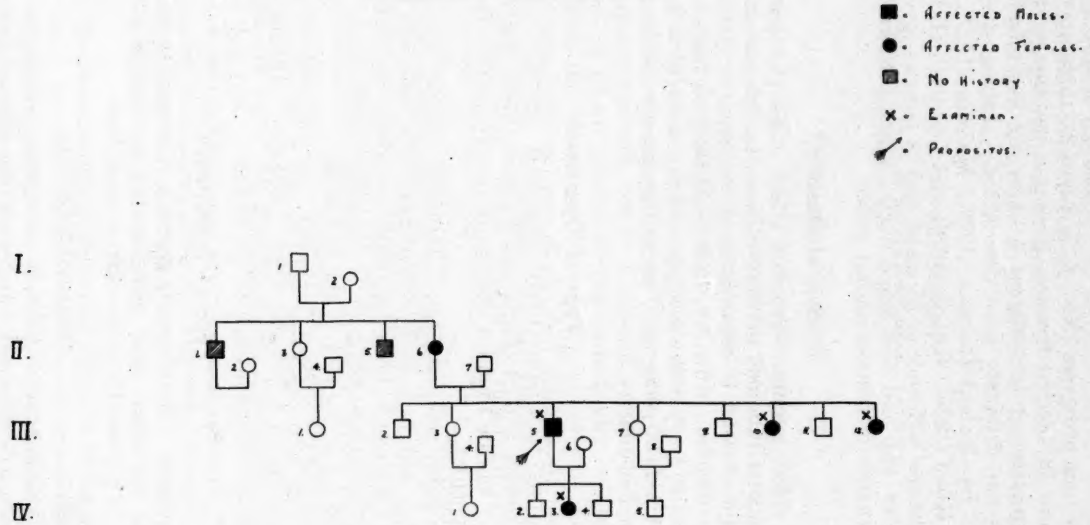
Type of Inheritance

In pedigree 7 the defect is possibly dominant in transmission, while in the other three pedigrees the number of affected individuals is too small to make even a conjecture.

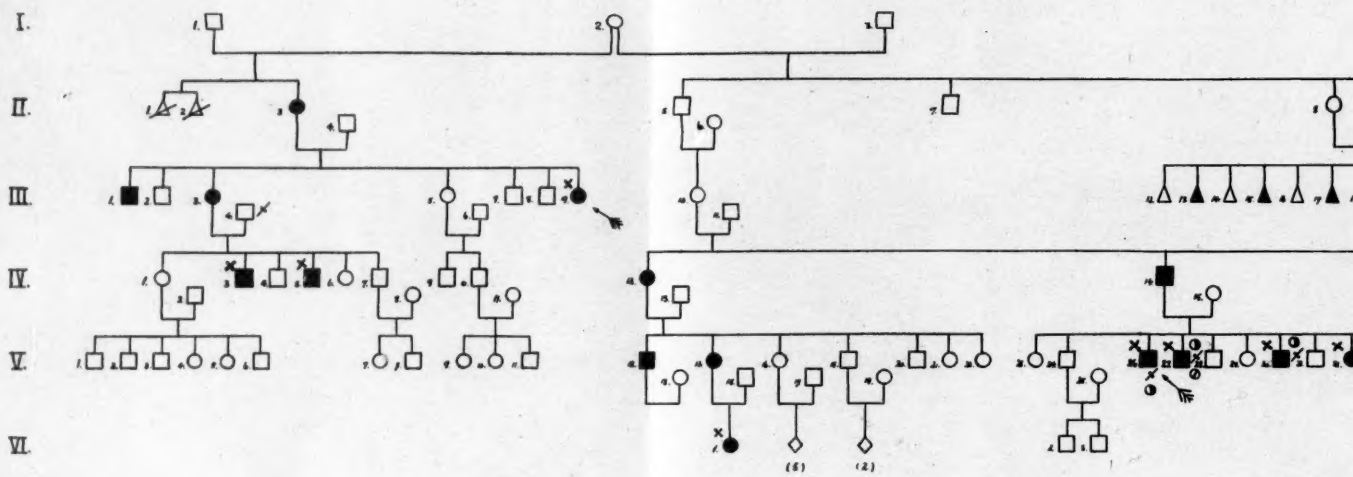
Associated Defects

Feeblemindedness.—There is no evidence of this in any of the four pedigrees—a small number of the affected individuals are somewhat retarded mentally and temperamental occasionally, but in no case could I classify them as feebleminded.

No. 6. CONGENITAL CATARACT.



No. 7. CATARACT.



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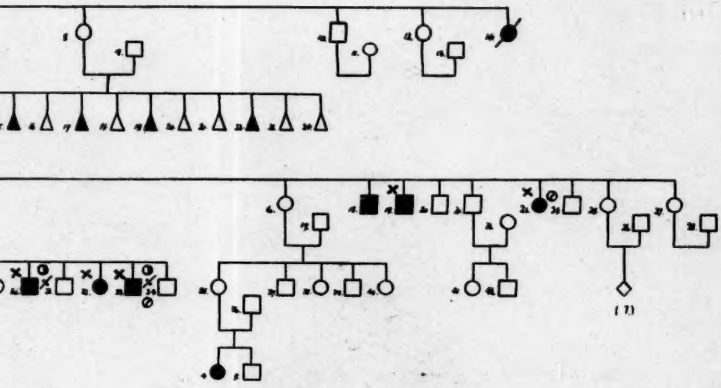

■ - Affected Males.
● - Affected Females.
✗ - Strabismus. - Convergent.
⊘ - Dislocated Lens.
◑ - Nystagmus.
△ - Sex Unknown.
✗ - Died in infancy.
× - Examined.
➶ - Propositus.

Dystrophia Myotonica.—The one pedigree (No. 10) of this disease is described separately.

Endocrine Disturbances.—There is no evidence of these in Tasmanian pedigrees.

Treatment

Operative interference in the form of repeated needling has been singularly successful in these Tasmanians, with one exception. When I say successful I refer to the anatomical, but not the functional results. All the poor functional results were due to late operation, and this stresses the fact that early operation is essential if good visual acuity is to be expected. To obtain this the public must be educated and its confidence gained.

Comments on Pedigrees

Pedigree No. 6.—This is an example of coralliform congenital cataract.

No. II/6, has cataracts but has not yet been examined.

No. III/5, aged 26 years, has had his right cataract needled after puberty, with a very fair visual result; while his left eye exhibits a typical coralliform cataract.

No. III/10 and 12—sisters of the above—have been examined by an ophthalmic surgeon, and certified for pension purposes as blind from congenital cataract.

No. IV/3, had typical coralliform cataract in a very advanced state right and left, with nystagmus at 13 months. Both cataracts have been needled, but it is too early yet to forecast the visual result.

Pedigree No. 7.—This pedigree is one of zonular cataract. III/9 is the eldest member examined, and also one of the propositi. She was examined at the age of 48 years. 25 years previously her right lens had been removed at operation, with good operative but poor visual results, the latter due to amblyopia. In her left eye besides her cataract she has gross keratoconus.

No. IV/5, has been examined for pension purposes, and certified blind from congenital cataracts.

No. IV/23, had opaque lenses right and left when examined by me at the age of 42 years. Both irides were tremulous, and the left lens obviously dislocated. There was marked nystagmus. I extracted the right lens by the intracapsular method of Sinclair, and although the anatomical result is good, acquired amblyopia prevents a good visual one. Nevertheless the vision is materially improved by the operation.

No. V/26, aged 23 years, has had both zonular cataracts needled by me, and his squint straightened. But nystagmus and amblyopia—both congenital, and ex anopsia prohibit a good visual result, yet he is considerably improved by the operative interference, and can use a typewriter with ease.

No. V/27, aged 21 years. Has also congenital cataracts, nystagmus, and squint. Both lenses are markedly dislocated, so that through the aphakic gaps with correction he is able to read 5/60 with both eyes together. In view of the post-operative visual result of his brothers, I feel that no operative interference would improve the present visual acuity.

No. V/30, aged 16 years. Had dense zonular cataracts right and left, with right convergent concomitant strabismus, and gross nystagmus. He has had both lenses needled, and his squint straightened by me three years ago; and is now pursuing a High School education. His visual result is poor due to amblyopia.

No. V/32 aged 12 years. Had only thick capsules right and left, although she had had no previous operations. A capsulotomy was done on each eye by me four years ago, and with correction she is able to see 6/36 right and left, and J. 10 right, and J. 16 left with bifocals.

No. V/33 aged 7 years, also had dislocated zonular cataracts, nystagmus and concomitant strabismus. Three years ago both lenses were needled, and his squint straightened by me, but the visual improvement is only slight owing to amblyopia.

I think V/32 certainly indicates that had these 5 sibs been operated on in infancy, the visual results would have been most satisfactory.

Pedigree No. 8.—No. III/36, aged 11 years, has had cataracts since birth, and was operated on when 5 months of age in both eyes, without result; and is now an inmate of a blind institution, with gross nystagmus, shrunken lenses, and secondary glaucoma.

No. III/23, aged 14 years, is placed in this order as he was examined a considerable time after III/36. He complained of defective sight and had marked anterior and early posterior cortical opacities in both eyes, but the vision with correction in each eye is still 6/12. I had been at a loss to account for the condition of III/36 until III/23 appeared.

Pedigree No. 9.—No. II/1, is a blind pensioner, certified by an ophthalmic surgeon as having congenital cataracts. The type is not stated, but his vision is less than 6/60 in each eye.

No. III/1, aged 19 years, has been examined by me, and has gross post-cortical lens opacities, so that right and left vision is reduced to hand movements. To date she has refused all operative interference.

Cataract with Dystrophia Myotonica

In 1911 Greenfield reported the first cases of cataract associated with dystrophia myotonica, and in 1924 Adie pointed out that cataract occurred alone in many individuals in a dystrophia myotonica pedigree. Vogt in 1921, and Goulden in 1928 gave a lengthy description of the slit-lamp appearance, so typical of the disease, and many cases of successful extraction have been reported (Souter, 1933). Rouquier and Chatain (1935) consider the neurological lesion to be in the diencephalon and the mesencephalon, but no satisfactory explanation has been given for the accompanying cataracts.

Comment on Pedigree No. 10.—I have but one pedigree (No. 10), to report, and that one exhibits cataract only in father I/1, and two children, II/8 and II/15, and dystrophia myotonica in the same two sibs. I can make little comment on I/1 who has not been examined by me, but has had a recent cataract extraction. II/8 had her left cataract extracted four years ago at the age of 37 years, and her right lens exhibits an advanced, but typical stage of myotonic cataract. She has obvious signs of muscle wasting and the typical myotonic grip. Her brother, II/15, conformed physically to an historic case reported by Caughey (1933), *viz.*:—" He is short, bald, speaking through his nose, looking forty at the age of twenty-five." As a matter of fact he was 29 when he came under observation, and had typical posterior star-shaped lens opacities in the cortex of both lenses. He had muscular weakness, and myotonic grip.

I needled the left cataract, and at present he has 6/4 and J. 1 vision in that eye, with aphakic correction.

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Chronic Glaucoma in Adults

Increased intra-ocular tension both in its acute and chronic form is relatively rare in Tasmania, and especially in its acute form. When chronic glaucoma does occur in the residents, it is surprising to find that a very marked proportion are not native born Australians. Out of 4,880 private case records during the past 5½ years (*i.e.*, January, 1931, to July, 1936),I can find the following cases of increased tension :—

Buphthalmos	1
Acute Glaucoma	5
Chronic Secondary Glaucoma	10
Chronic Primary Glaucoma	44

If an analysis is made of my 4,880 cases, then the percentage of chronic primary glaucoma amongst these cases, is 0·9 per cent., which compares favourably with the statistics quoted by Julia Bell (1932) which ranged from 0·4 per cent. in Germany to 2·73 per cent. in Italy; and estimates from almost a million European cases make an average percentage of 0·97 per cent.

Of these 44 cases of chronic primary glaucoma, 30 were native born Australians, and 14 were born in the British Isles, which in percentages is 68·2 per cent. and 31·8 per cent. respectively.

Now, according to the 1933 Census, 95 per cent. of the population of Tasmania were born within the Commonwealth, and 5 per cent. outside the Commonwealth, so that one would have expected to find the percentage of glaucoma identical. Presuming 5 per cent. of the population are not native born, then 244 of the above 4,880 cases should have been born outside Australia, and 4,636 of the cases born within Australia. Of these approximately 244 cases, no less than 14 have glaucoma—a surprisingly high percentage of 5·73, while of the 4,636 Australian born cases, only 30 have glaucoma—a surprisingly low percentage of 0·65. I am unable to account for these figures, and intend reviewing them again at a later date, in order to confirm the startling disproportions. If we conclude that " worry " is a predisposing cause of glaucoma, then a faint ray of truth may be revealed.

The recent emigrants have undoubtedly been subjected to far more anxiety than their Tasmanian brothers and sisters, who by reason of the prolonged isolation of Tasmania, and the isolated position of their residences were prohibited reasonable contact with the outside world until 1925. Since the advent of radio in 1925, and the world-wide telephone communication in 1936, this isolation to a great extent has been banished, and it may be interesting to see if the glaucoma figures will rise in the next half of this century (See Table VI).

Hereditary Influences

Of my 44 cases of chronic primary glaucoma, only one hereditary pedigree (No. 13) of 2 cases can be found. The two cases of pedigree 14 are amongst these 44 cases, but this is purely familial in incidence, but I think worth quoting. Pedigree 11 was given me by a patient who had not glaucoma herself, while No. 12 was kindly supplied by Dr. Counsell. So that amongst my own patients the hereditary factor is extremely low. Bell (1932) complains of the paucity of reliable records of hereditary glaucoma, and finds only 3 pedigrees reported by Nettleship, and one by Usher, and was able to collect only 68 reported pedigrees in the literature.

Sex Incidence of Hereditary Glaucoma

Taking the 3 hereditary pedigrees (Nos. 11, 12 and 13), I find in a total of 8 cases there are 5 males (62·5 per cent.) and 3 females (37·5 per cent.) in all. Whereas Bell's figures (1932) are 99 males to 97 females. Zorab (1932) on the other hand in surveying 6 pedigrees found 25 males and 10 females affected; while Frank Kamenetzki (1925) recorded a pedigree in which every affected member was a male.

So that there appears to be no doubt that males are more prone to be affected than females, when the disease is hereditary. Conversely, of the 42 non-hereditary cases seen by me since 1930, 25 were females (59·5 per cent.), and 17 males (40·5 per cent.), which gives a greater predominance of females, and conforms to Rimpler's statistics—quoted by Frank Kamenetzki—of 2,021 cases of glaucoma in which 1,150 (56·9 per cent.) were females, and 871 (43·1 per cent.) were males.

Age of Onset

I can give no useful information as to this. Pedigree 11, which was collected long before I anticipated writing this paper, might have helped considerably, but these details are wanting unfortunately, and quite possibly the propositus would not have been able to supply them. In my own pedigree (No. 13) one patient was 88, and his niece 37; while in the familial pedigree (No. 45) the two cases examined were 86 and 84 respectively.

Mode of Transmission

Although Macklin (1927) considers the transmission of hereditary glaucoma dominant, yet my few pedigrees prove that this is not always so by any means, and even so great an authority as Nettleship did not feel disposed to commit himself on the mode of transmission.

Frank Kamenetzki (1925) reports one pedigree in which all the afflicted members are males, and the mode of transmission was a recessive sex-linked one.

Concurrent Anomalies

In the very interesting pedigree of Frank Kamenetzki (1925) the individuals affected with glaucoma had an accompanying atrophy of the iris stroma. Bell (1932) states, " So far as I can judge, cases of hereditary glaucoma appear in otherwise healthy stock, and very few examples of associated hereditary diseases or anomalies are found amongst the affected individuals." My few cases exhibit no peculiar anomalies.

Treatment

There appears to be little variation from the orthodox treatment in the hereditary cases. Frank Kamenetzki (1925) found iridectomies difficult in his cases owing to the iris atrophy, while James (1927) reports satisfactory results from trephining. Personally I have found no indication in my few cases to depart from routine procedures.

Comment on Pedigrees

Pedigree No. 11.—As mentioned before this pedigree was obtained roughly, long before I intended to write this monograph, and therefore is not very helpful. The propositus III/5, called to be examined on account of the predominance of glaucoma in her family. She herself was unaffected, and as the other members of her family live, or lived in the north of the island, I have been unable to examine them to date.

Pedigree No. 12.—Was kindly supplied to me by Dr. Counsell. I/1 is dead, while II/1, at the age of 70 has had to have one eye trephined.

Pedigree No. 13.—III/3, aged 88 years, is under treatment by me for cavernous atrophy of the optic discs, and raised tension—central vision has been lost in one eye, but the fields of vision are normal to 1° white. His niece IV/17 has advanced glaucoma right and left at the age of 37 years. Four other members of the family examined are free from this defect, but all four of these exhibited gross astigmatism in both eyes.

Pedigree No. 14, are 3 sibs. II/4 is dead, but his 2 sisters (II/1 and 3) at the age of 89 and 86 respectively, have advanced chronic glaucoma in both eyes, and they did not report for treatment until aged 86 and 84 respectively. One significant fact about these old ladies is that when each reported (not under miotics)

the tension in both eyes was 20 mm. Hg. (Schiötz), *i.e*, the four eyes had similar normal tension, and yet the four fields were greatly, and typically constricted; and the optic discs grossly cupped.

TABLE VI

Incidence of Hereditary Glaucoma in Tasmania from January 1, 1930, to July 1, 1936.

No. of cases examined	4880
Chronic Primary Glaucoma	44 = 0·9 per cent.
Born in Australia	30 = 68·2 per cent.
Born outside Australia	14 = 31·8 per cent.
Males	18 = 40·9 per cent.
Females	26 = 59·1 per cent.
Hereditary cases amongst above	2
Males	1
Females	1
Non-hereditary cases amongst above	42
Males	17 = 40·5 per cent.
Females	25 = 59·5 per cent.
No. of hereditary cases reported in Tasmania	8
Males	5 = 62·5 per cent.
Females	3 = 33·5 per cent.
Percentage of native born Australians in population	= 95 per cent.
Percentage of non native born Australians in population	= 5 per cent.
Approximate number of my 4880 cases born in Australia	= 4636
Approximate number of my 4880 cases born outside Australia	= 244
Percentage of glaucoma amongst 4636 assumed Australian born cases	= 0·65 per cent.
Percentage of glaucoma amongst 244 assumed non-Australian born cases	= 5·75 per cent.

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Keratoconus

Although keratoconus is an hereditary disease, yet, as in retinitis pigmentosa, many sporadic cases, in which there appears to be no hereditary influence whatsoever are found in routine work. In my 4,880 private cases (mentioned under glaucoma and pterygium) I find 19 cases in all of keratoconus—making a case incidence of 0·39 per cent. Of these 19 cases, only one can definitely be said to be hereditary (pedigree 16), while three others are strongly familial in incidence (pedigree 15). Of these 19 cases, 18 came from Southern Tasmania where the proportion of city and country dwellers is equal. Yet of these 18 patients no less than 14 came from the country districts, where good food and sunlight exist in abundance.

Associated Defects

Fleischer's Ring.—This was present in only one of the 38 eyes examined and was not accompanied by opacity of the cornea.

Apical Corneal Opacities.—These were present in 6 of the 38 eyes, and in 2 eyes they received surgical treatment.

Mode of Transmission

According to Franceschetti (1930), keratoconus is transmitted both as a dominant and recessive. In my one pedigree (No. 24) we must undoubtedly presume that the mode of transmission was recessive.

Sex Incidence

Of these 19 patients, 12 were females, and 7 males, while the one hereditary pedigree (No. 16) shows only females affected. As I am unable to obtain a copy of Jaonsch and Stahli's articles, I cannot compare and contrast these figures with statistics from other countries.

Concurrent Diseases

Mongolian Idiocy.—Two of these 19 patients were Mongolian idiots, with advanced conical cornea in 3 eyes, which needed and received operative interference.

Thyroid Dysfunction.—Of the 19 patients, 10 were generally examined. Three showed no trace of endocrine dysfunction, three females were subthyroid, and 4 hyperthyroid. Knapp (1924) found low basal metabolic rates in 2 out of 6 patients.

Treatment

Contact Lenses.—These were prescribed in one case, and 2 other cases had their visual acuity tested with them. In all three cases the result was highly satisfactory. But a problem, which needs further investigation and discussion, is the possibility of retarding the progress of the corneal bulging by ordering contact lenses early in the disease.

Miotics and Hormone Therapy.—Although I have given both these prolonged trial, I frankly admit that I have seen very little benefit derived from their use. In one case I thought "hormotone" retarded the progress.

Surgical Interference.—This was necessary on 3 eyes, and the electric cautery was applied to the apex of the cones until perforation resulted, after the manner of Knapp (1929). In one case the anterior chamber took three weeks to reform but the ultimate results in all were satisfactory.

Comments on Pedigrees

Pedigree No. 15.—This is purely familial in origin, but it is worthy of record, and reports four members of a sibship of 11, with marked keratoconus. All four of the affected members and two of the unaffected members were examined by me.

II/2, aged 37 years, cornea normal.

II/6, aged 34 years. Gross keratoconus right and left without corneal scarring. Right and left vision improved to 6/12 with high oblique concave cylinders.

II/10. Gross keratoconus. No details.

II/11. Normal cornea.

II/12, aged 24 years. Early keratoconus right and left. Vision right and left improved to 6/12 with myopic correction.

II/15, aged 19 years. Moderate keratoconus right and left without scarring. Keratoconus more marked in right eye.

III/9. Has defective eye sight. Doubtful causation.

Pedigree No. 16.—III/2, aged 20 years. Was examined by me, and has gross keratoconus right and left, but no corneal opacities.

III/8. A cousin was examined by Dr. Homewood of Launceston, and found to have keratoconus also, but I have no details.

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Microphthalmos

Among the hereditary eye diseases microphthalmos stands prominent as a potent cause of blindness. Bell (1932) analysed the vision of 91 cases of microphthalmos and found total blindness in both eyes of 33 cases, and vision of 6/60 or less in both eyes of 25 cases; blindness in one eye of 8 cases; " very bad " bilateral vision in 10 cases, leaving only 15 cases with good vision in both eyes, and even 4 of these 15 have latterly developed cataracts. She also analysed the other ocular defects accompanying microphthalmos, and pointed out the predominance of iris and lens defects reported. Usher (1921) has especially drawn attention to accompanying corectopia and myopia when reporting a pedigree of 11 affected individuals in five generations. Bell was unable to elucidate any peculiarities about sex incidence, and inheritance of microphthalmos; but on the other hand Ash (1922) has reported a pedigree of 34 individuals in four generations, eleven of whom were affected with microphthalmos, and every affected one was a male. Like Leber's optic atrophy the disease in this pedigree was strictly sex linked, and was transmitted by unaffected females only. The significance of this pedigree has been elaborated by Macklin (1927). In her 41 pedigrees Bell records consanguinity only 5 times. Wolff (1930) on the other hand considers consanguinity precipitated his pedigree, in which 5 of 10 children were afflicted with microphthalmos. (I have made reference to a similar occurrence under Leber's Disease.)

Treatment

I have no comment to offer except that great cosmetic improvement resulted from ordering 10·0 sphere before the left eye of V/5, for which the parent was most grateful.

Comments on Pedigrees

Pedigree No. 17.—This was given me by the mother (IV/9) of my patient (V/4), who was intelligent and most co-operative. She went to great pains to obtain it, and I think I can vouch for her integrity. I have been unable to examine IV/32 and so will briefly describe V/4's condition observed at annual examinations over 4½ years.

Right Eye.—Normal size. Dense crescentic corneal opacity below, extending inwards from the limbus, pupil circular and not ectopic. Gross pseudoneuritis.

Right vision under mydriatic with

$$\begin{matrix} +\ 2{\cdot}25 \text{ D. sph.} \\ \hline +\ 3{\cdot}25 \text{ 105} \end{matrix} = 6/6 \text{ in all and J. 1.}$$

Left Eye.—Very small. Dense crescentic corneal opacity below, extending inwards from the limbus. Corectopia downwards. Opaque lens dislocated upward, giving faint fundus reflex below. No fundus details with the pupil dilated.

Left vision = Hand Movements.

V/4's only sister (V/5) has " port wine stain " of the face, but both her eyes are normal.

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Myopia

According to Best (1934) myopia is the cause of 0·3 per cent. of blindness in the U.S.A., but just how prevalent it is in the community is not stated. However, in my 4,880 private cases there were 641 cases of myopia and myopic astigmatism; which gives a case incidence of 13·13 per cent. In Counsell's and my analysis of the 170 blind in Tasmania we found that myopia would account for 4 out of 92 with reliable information, *i.e.*, 4·35 per cent., which compared with Best's figures above, is very high.

That myopia is undoubtedly hereditary is shown by my 9 pedigrees (Nos. 18 to 26), but some authors—Levinsohn (1934), and Franceschetti (1930)—seriously doubt the hereditary factor, while Roden (1933) tries to explain all cases on a mechanical or pathological basis. At least 4·5 per cent. of my 641 myopes gave an hereditary history.

Mode of Transmission

Holm (1925) considers the mode of transmission may be dominant, or recessive, and this is borne out by my 9 pedigrees, although most other authors consider it recessive only in transmission.

Sex Incidence

Of 48 affected persons mentioned in my 8 pedigrees, 21 were males, and 27 females; giving a sex incidence of 44·7 per cent., and 55·3 per cent. respectively. I am unable to find figures in the literature for comparison, but Wilson (1935) considers 2/3 of myopes are females.

Associated Defects

Night Blindness.—Although reported by Holm (1925), there is no evidence of it in my pedigrees.

Exophoria.—This was only exhibited to any degree in one of the 29 cases examined, and therefore cannot be an important factor in the production of hereditary myopia, as suggested by Pascal (1935).

Treatment

To me the main interest lies in treatment. We are all conversant with the necessity for adequate correction, and limited close work, and we are all aware of the disappointing results. As the theory of calcium deficiency has been advocated by many writers, I had the blood calcium in six progressive myopes examined, with only one subnormal result. It was then that my colleague, Dr. A. W. Shugg, suggested that perhaps progressive myopia was not a matter of calcium deficiency, but one of defective calcium utilisation. Acting on his advice, I have 22 patients on " radiostoleum " (vitamin A and D), and the first 2 which were progressing rapidly over an 18 months' period, ceased progress at once, and have remained stationary since. The other 20 cases have been under treatment for too short a period to comment upon, but I intend to report the results later. Walker (1932) reports favourable results from calcium and parathyroid; but admits failure from calcium therapy alone, while Law (1934) reported satisfactory results from calcium and parathyroid, and D. V. Giri who entered into the subsequent discussion suggested that vitamin D instead of parathyroid might aid the assimilation of calcium. This fact I am endeavouring to affirm, and to date the result has been more than encouraging.

Comments on Pedigrees

Pedigree No. 18.—I/1. Believed to be normal.

I/2. Not examined, but supposed to be myopic.

II/1, aged 14 years. Had more than 17 dioptres of myopia right and left, but was not refracted.

II/2, aged 12 years. High compound myopic astigmatism. Now on radiostoleum.

II/3. Not examined, and believed to be normal.

II/4, aged 7 years. Lamellar cataracts right and left, of the tetany type. Moderate simple myopic astigmatism.

This family is of extreme interest. I/1 was a lighthouse keeper on a most isolated and wind-swept island on the southern coast

of Tasmania. Fresh fruit and vegetables, also fresh milk were unprocurable for a greater part of the year, and so the children's calcium intake was very low. As the children arrived at school age they were tutored by correspondence course until considered sufficiently responsible to send to a city school. II/4 was still dwelling on the island. Apparently her calcium metabolism had been so deranged in early life that cataracts resulted, and I think the presence of these cataracts in conjunction with high myopia in her sister and brother, definitely point to a faulty calcium metabolism as an aetiological factor in myopia.

Pedigree No. 19.—III/4, aged 20 years. Compound myopic astigmatism of over 20 dioptres with definite choroidal stretching, and one haemorrhage.

III/5. Amblyopia ex anopsia as the result of squint in childhood. Eyes now straight by cover test.

III/7, aged 25 years. First cousin of III/4. Has low grade compound myopic astigmatism.

Pedigree No. 20.—I/1. Believed to be myopic, and certainly has defective sight.

II/1, aged 44 years. High compound myopic astigmatism, with the rule, with choroidal stretching.

II/3, aged 40 years. Right retinal detachment, cured by Larsson method. High compound myopic astigmatism with the rule, with albinism and nystagmus.

II/7, aged 34 years. Simple myopic astigmatism with the rule.

II/11, aged 24 years. High myopic astigmatism with the rule.

III/4. High mixed astigmatism with the rule.

It is interesting to note that the astigmatism in this pedigree is with the rule in every case.

Pedigree No. 21.—III/8, aged 85 years (1932). Simple myopic astigmatism against the rule—Ocular palsy—cavernous atrophy of discs.

III/10, aged 84 years (1935). High compound oblique myopic astigmatism in right eye. Gross lens and fundus changes in left eye.

III/12, aged 82 years (1936). Compound hypermetropic astigmatism with cavernous atrophy of both discs, accompanied by right central scotoma.

III/13, aged 47 years (1910). Myopic astigmatism against the rule. Wife of III/12.

IV/4. Hypermetropic astigmatism. Also epilepsy.

IV/6, aged 61 years (1936). Compound hypermetropic astigmatism with the rule.

IV/7, aged 55 years (1932). Wife of IV/6. Compound hypermetropic astigmatism.

IV/10, aged 57 years (1933). Compound myopic astigmatism against the rule.

IV/11, aged 58 years (1931). Hypermetropia. Wife of IV/10. Died from 8th nerve tumour.

IV/14, aged 51 years (1936). Oblique compound myopic astigmatism.

IV/18, aged 53 years (1934). High oblique compound myopic astigmatism.

IV/20, aged 22 years (1906). Compound myopic astigmatism against the rule. Died of pulmonary tuberculosis.

IV/22, aged 58 years (1935). Compound myopic astigmatism against the rule.

IV/23, aged 54 years (1935). Compound myopic astigmatism against the rule.

IV/24, aged 53 years (1935). Compound myopic astigmatism against the rule.

IV/27. Emmetropic.

IV/29. Emmetropic.

IV/32, aged 51 years (1935). Compound myopic astigmatism against the rule.

IV/33. Presenile cataracts. Wife of IV/32.

IV/34, aged 48 years (1934). Pure myopia.

IV/37, aged 45 years (1931). Oblique compound hypermetropic astigmatism.

IV/42, aged 48 years (1932). Oblique compound hypermetropic astigmatism.

V/9, aged 35 years (1936). Hypermetropic astigmatism with the rule.

V/21, aged 16 years (1933). Oblique myopic astigmatism.

V/25, aged 26 years (1935). Mixed astigmatism.

V/32, aged 17 years (1933). Myopic astigmatism against the rule.

V/42, aged 15 years (1931). Pure myopia.

This pedigree seems to conform to both dominant and recessive types of transmission, but as many remain to be examined, one cannot be too dogmatic. Of the 13 cases of astigmatism no less than 8 were against the rule, which is significant.

IV/34 and V/42 are interesting, as father and daughter were the only examples of pure myopia in the pedigree.

Pedigree No. 22.—III/1. Low compound myopic astigmatism.

IV/1. Not examined.

IV/2. Low compound myopic astigmatism. Refraction very similar to father—III/1.

IV/7. High compound myopic astigmatism.

Pedigree No. 23.—I/2. Not examined.

II/3. High compound myopic astigmatism, with old choroiditis and positive W. R.

II/7. Not examined.

III/2. Moderate, compound myopic astigmatism.

The myopia in this pedigree is certainly dominant. 1/2 transmitted it to half her children, and so did II/3.

Pedigree No. 24.—II/1 and 2. No details.

III/6, aged 17 years. High compound myopic astigmatism of over 20 dioptres.

III/7, aged 14 years. High compound myopic astigmatism of over 10 dioptres.

Pedigree No. 25.—I/1. Normal.

II/3. Compound myopic astigmatism with the rule.

II/4. Compound myopic astigmatism with the rule. Stammers.

II/7. Compound myopic astigmatism with the rule.

II/8. Compound myopic astigmatism with the rule.

Examination of I/2 may be the clue to the mode of transmission.

Pedigree No. 26.—IV/2, aged 20 years. High myopia without astigmatism.

It is surprising that more members of this sibship do not as yet show myopia, in view of evidence of the disease in both paternal and maternal relatives.

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Nystagmus—Consanguinity

The spasmodic precipitation of hereditary eye disease had always remained a mystery to me, until I commenced to collect pedigrees for this paper. Then the pernicious influence of consanguinity became apparent, and will be referred to again under Leber's optic atrophy, and especially under retinitis pigmentosa.

In my pedigrees on nystagmus, consanguinity is evident both in pedigrees 4 and 30. It will be seen in pedigree 4 that in generation III two first cousins married in a family in which hereditary nystagmus was recessive, and as the result 3 of the 4

sibs of this union developed the disease in a gross form. In the case of pedigree 30, repeated consanguinity precipitated the first case of nystagmus in a perfectly healthy family. Many branches of this latter family are personally known and have been examined by me; and it is surprising, with the frequent inter-marrying which has taken place, that more members have not exhibited congenital defects in one form or another.

Mode of Transmission

Hereditary nystagmus may be transmitted in one of two ways:

(*a*) Ambisexual group, where either parent transmits it to both sexes.

(*b*) Recessive male limited group, transmitted by unaffected females to their male offsprings.

According to Knighton (1929) and Holm (1927) (*b*) method of transmission is rare. Pedigree No. 27 in which only males could be affected, will be watched with interest by me for evidence of sex-linkage.

Sex Incidence

In my 4 pedigrees, the sex incidence was 9 males to 3 females—*i.e.*, 75 per cent. males. I therefore took 5 available reported pedigrees, at random, and found that the total sex incidence in these was 42 males to 13 females, *i.e.*, 76·36 per cent. males. This is a very interesting comparison, and I think proves that the sex incidence in Tasmania conforms very closely to that in other parts of the world.

Concurrent Defects

Albinism.—Partial albinism is most apparent in nearly all cases of congenital nystagmus, and pedigrees 29 and 4 exhibit this tendency most markedly.

Head Nodding.—This has been reported by Cox (1936), and many other authors, but I have not found a case of this in my pedigrees. It must not however, be confused with lateral rotation of the head to one side, and both eyes to the other, which many patients take up in order to steady their eyes, and improve their visual acuity. Case III/81 of pedigree 4 had developed this habit to a marked degree. Nor must this head nodding be confused with spasmus nutans—a totally different clinical entity.

Astigmatism.—This is a most common feature as indicated by Holm (1927), and Niccol (1915), and its correction offers one hope

in the alleviation of this disease. Frank (1936) states it is the only treatment of any account.

Amblyopia.—This varies considerably in members of the same pedigree. Pedigree No. 27 has 3 affected members, I/1 and II/2 having 6/9 or better vision with correction; while II/1 would not improve above 6/24 in each eye. Similarly in pedigree 4, IV/18 would not improve above 6/60 right and left, while III/81 improved to 6/9 partly with astigmatic correction. It is interesting to note here, that Usher in 1912 (quoted by Niccol) reported a case of congenital nystagmus in which "a microscopic examination of the retina showed that there was no proper fovea and a few ganglion cells were present in this region. There was no evidence of lack of retinal pigment."

Stammering.—Although none of the affected members of pedigree 4 was affected with stammering, yet two brothers of III/81 had this defect in a marked degree.

Treatment

Except for correction of refractive errors, and especially astigmatism, there is little treatment availing. But I must stress here, that careful retinoscopy combined with careful mydriatic and post-mydriatic subjective testing will frequently produce quite promising results, and due compensation for extra care taken. Orthoptic training which has been mooted on occasions, appears to hold out definite hope of improvement. III/81 has responded rapidly to it.

Comments on Pedigrees

Pedigree No. 27.—The father I/1 has high myopic astigmatism, the correction of which produces 6/6 in either eye. He also has 30° of alternating divergent concomitant strabismus, while both fundi are absolutely normal. His eldest son (II/1) aged 12 years, has hypermetropic astigmatism, but his vision will not improve above 6/24 in either eye. His brother (II/2) aged 10 years, on the other hand is practically emmetropic, and has 6/9 in each eye. Both sibs have normal fundi.

With regard to the nystagmus in this family, I/1 and II/2, who have good central vision, have only nystagmus when both eyes are deviated to one or the other side; while II/1 with poor central vision has his nystagmus most marked when looking straight ahead.

Pedigree No. 28.—Both affected members (I/2 and II/1) have fine horizontal nystagmus. While I/2, aged 36 years, has pure

myopia, and II/1, aged 9 years, has hypermetropic astigmatism—all four eyes correct to 6/9 all.

Pedigree No. 29.—The one examined member (IV/2), aged 10 years, has been under my observation for 6 years. He is partial albino, has gross mixed astigmatism, and with correction improves to 6/18. The nystagmus is more marked on looking laterally, and both maculae exhibit fine pigmentary stippling in contrast to the pale remainder of the retina.

No. 4 (See Astigmatism).—This complex pedigree has been referred to before under astigmatism. I shall here refer only to the right hand section of the pedigree. III/60 and 63 have divergent squints and ambylopia, but have been dealt with under astigmatism.

III/81, aged 13 years, the most recent member under observation, has marked nystagmus, with mixed astigmatism, the correction of which improves her vision from 6/24 to 6/12 each eye.

III/79 and 82. No nystagmus, but stammering.

IV/18, aged 19 years. Has high myopic astigmatism and nystagmus, with only 6/60 in each eye. His brother and sister, although not examined, I believe have grossly defective sight also.

IV/22 is interesting. Although he has no nystagmus, he has congenital ambylopia of his right eye, without any lateral deviation, and only 5 dioptres of vertical deviation.

IV/26. Has not been examined.

Pedigree No. 30.—This has been included to show how repeated consanguinity will precipitate congenital nystagmus. IV/2, aged 47 years, and V/1, aged 22 years, have been examined—the former is almost emmetropic and shows no nystagmus, while V/1, as well as the nystagmus, has high myopic astigmatism, and will not improve beyond 6/24 in either eye.

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(To be concluded)

A SIMPLE ACCESSORY FOR THE DARKROOM

BY

J. SHERNE, F.R.C.S.

LEEDS

IN view of the interest shown in this little aid to the darkroom at the recent meeting of the North of England Ophthalmological Society, and as it did not figure in the programme, I thought it might be worth while introducing the idea to a larger audience.

On the darkroom wall, directly opposite to and on the same level as the retinoscopy light, is fixed a reflector such as cyclists use instead of a rear light. This picks up the light extraordinarily well, producing a brilliant red fixation spot, which even the most myopic seem to have no difficulty in seeing.

I now do the great majority of hospital refractions without a mydriatic as I feel that under proper conditions it is the most accurate method, and I have found this method a great advance on the other methods I have tried of persuading the patient to relax his accommodation.

In refracting the right eye I sit in front of and slightly to the left of the patient so that his view of the reflector by the right eye is obstructed by my head, but he has a clear view with the left eye. He is now asked to keep looking at the red light, so that when I am refracting the right eye, his direction of gaze is straight forward, fixing the opposite wall of the darkroom.

For refracting the left eye I shift my stool slightly to my right, so that my head now obstructs his left eye, and he fixes the reflector with his right eye. This involves using the left eye and hand for the retinoscopy, holding the lenses in the right hand. After a little practice this is not very difficult.

These reflectors are very cheap, and can be bought for a shilling at Halford's or any other cycle store. They are very easy to fix on the wall and can readily be adjusted to any height. Children seem to be more interested in them than in an ordinary spotlight and no doubt other uses for such a simple device will occur to readers.

FIFTEENTH INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

THE fifteenth quadrennial Congress was held in Cairo from December 8 to 14 under the presidency of Professor Nordenson of Stockholm. The first morning was occupied by the ceremonial opening of the Congress in the presence of His Majesty King Farouk. This took place in the large hall of the Egyptian

University and was well attended by most of the 600 congressists and the 200 associate members. Speeches were delivered by the national representatives, Dr. Sinclair of Edinburgh representing Great Britain.

In the evening there was a Reception at the Semiramis Hotel by the Local Committee, during which there was a cabaret entertainment followed by a buffet supper. On the following day, Thursday, the congressists were honoured by invitation to a reception at the Palace of Abdine by command of His Majesty. The delegates from the various countries were presented. An admirable play was given in the Palace Theatre in Arabic, the heroine of which was portrayed as being blind. After this there was a ballet by the professional ladies of the Opera House, and then excerpts from the opera, "The Barber of Seville." It should be mentioned that the orchestra was extremely good, the members of which came from the State Opera House. The King was present throughout the entertainment, and at the supper.

On Friday evening the congressists and their consorts were invited to a banquet given by the Minister of Public Health. The admirable cuisine, the wines and the attendance by the Berberine waiters are to be remembered; but Shepheard's Hotel has always been celebrated for its Lucullus feasts.

On Saturday afternoon there was an excursion to see the Pyramids and the Sphinx, with tea at Mena House Hotel. On the same evening there was the official banquet at the Semiramis Hotel. On Sunday morning a visit was paid to the Memorial Ophthalmic Laboratory; this is probably the finest ophthalmic laboratory in the world, as to architecture, design and equipment with every known ophthalmic instrument of the latest type; the arrangements for keeping the monkeys and other animals used for experimental research were especially admired. On Monday there was a visit to one of the newer ophthalmic hospitals at Rod-el-Farag, which has been built at a cost of £28,000, in the environs of Cairo. This was followed by a steamer excursion to the Delta Barrage and the wonderful gardens in the vicinity. On Tuesday one of the original travelling hospitals was visited; this commenced work in 1904, though the general equipment and amenities have been greatly improved since then, all modern ophthalmic instruments being now available.

On Monday evening there was a reception by the Minister of Public Health at the Semiramis Hotel with a buffet supper. On Tuesday afternoon there was a General Meeting at which the place for the next quadrennial Congress was decided. The suggestions were for Vienna, Warsaw and New York, the first of which was decided on. The resignation of Sir John Parsons from membership of the International Council was accepted with regret and

Dr. Sinclair, a former President of the Ophthalmological Society of the United Kingdom was appointed in his place. Two new *ex-officio* members of the International Council were appointed, Mr. MacCallan as President of the International Organisation against Trachoma, and Dr. Bailliart as President of the International Association for the Prevention of Blindness. Professor Nordenson was unanimously re-elected President of the International Council for another four years.

Every effort was made by the Egyptian members of the Local Committee to provide for the pleasure and convenience of congressists. Delightfully sunny weather was experienced during the whole of the Congress. Numbers of the congressists journeyed to Luxor and Asswan, while every one made several visits to the entrancingly interesting museums of Cairo. At no Congress has greater pleasure and interest been experienced by those who attended it.

At the scientific meetings the two main subjects discussed were:

(1) Hypertension and retinal arterio-sclerosis and
(2) Endocrinology and its relation to the eye.

Dr. H. P. Wagener and Dr. Norman M. Keith (the Mayo Clinic), Dr. Bailliart (Paris) and Dr. Y. Koyanagi (Japan) read the opening papers on the subject of retinal arterial hypertension.

The ophthalmologist, because of the wider and more intimate knowledge of the clinical details of retinal pathology, should be better equipped than the physician to observe the reactions in the retina which are associated with an elevation of blood pressure and systemic vascular disease. If it can be assumed, as seems logical, that the visible reactions in the retinal vessels are similar, though at times disproportionate, to the invisible reactions taking place in vessels of similar size throughout the body, it is obvious that an explanation of the mechanism of these reactions in the retina will go far toward solving the mechanism of diffuse vascular disease. The responsibility of the ophthalmologist in the solution of this problem is great, and his opportunities are wide, for he is able to see these vascular changes from their inception and to study their modes of origin and progression.

Evidence accumulated in the fields of physiology, pathology and clinical investigation seems to indicate rather conclusively that arterial hypertension is an expression of a diffuse disease or abnormal condition of the arterioles throughout the body. The lesion of the arterioles results in an increase in the peripheral resistance in certain regions or in the whole of the arteriolar bed. The narrowing of the lumen of the arterioles, which is the apparent cause of the increased resistance is not structural in the earliest

phases of the disease. It must be regarded as due to increased tonicity of the vessel wall, to spastic constriction, or to a combination of the two. Whether the tonic or spastic constriction is due to the action of a pressor substance directly on the arteriolar wall or through the medium of the sympathetic nerves, or simply to increased vasomotor stimuli is not known. If the causative factor of the arteriolar narrowing continues to operate, histologic changes develop ultimately in various parts of the arteriolar system, for example, in the peripheral muscles, the heart, the kidney, the brain and the retina.

Both these factors, functional narrowing of and structural wall changes in arterioles, appear to be present in the usual case of diffuse arteriolar disease of the so-called "essential hypertension" type. On the basis largely of the dominance of one or the other of these factors in a particular case, it is possible to classify cases of "essential" hypertension into various groups, in each of which the probable course of the disease and the life expectancy of the individual patient can be foretold with a reasonable degree of accuracy.

Dumas divided essential hypertension into three phases: In the first or silent phase the patient is essentially symptomless. In the second or established phase the disease has become organic and visceral lesions are present. Cerebral haemorrhages, cardiac failure or uraemia may terminate this phase. It is possible, however, for the hypertension to pass into the third or involutional phase, in which the blood pressure drops and arteriosclerosis dominates the picture—the hypotensive stage of hypertension. According to Bonamour, the retinal changes in Dumas' first phase are spastic in type. The blood pressure in the central artery of the retina is likely to be elevated disproportionately to the blood pressure in the brachial arteries. Patients may complain of transient loss of vision. Post-spastic thrombosis of the central artery may result in permanent loss of vision. In the second or established phase, retinitis develops in cases which are running a rapidly progressive course. If the disease progresses more slowly, sclerosis develops in the retinal arteries as a defence reaction. In the third or involutional phase, the sclerosis in the retinal arteries is likely to be quite marked, and lesions such as arteriosclerotic retinitis and retinitis circinata are characteristic. In Bonamour's opinion, retinitis of the hypertensive type is essentially vasospastic in origin. As a result of arteriolar constriction reactional dilation and stasis develop in the capillary bed and serous and haemorrhagic transudations take place into the retina.

The problem of the pathogenesis of "albuminuric retinitis" has been a consuming one almost since the beginnings of our

knowledge of albuminuria as an expression of disease of the kidneys. Bright himself recognised that loss of vision was a symptom in cases of certain patients with albuminuria.

Most physicians at present agree that a retinitis of "albuminuric type" can occur in cases in which no evidence of renal insufficiency can be demonstrated clinically, and in which, at necropsy, the kidneys show no evidence of primary nephritis.

The most logical explanation of the mode of origin of "albuminuric retinitis" seems to be that of Volhard, who regarded both the arteriolar sclerosis of Verwey and the retinitis as a result of ischaemia due to spasm in the larger arterioles. Volhard's conception of ischaemic or angiospastic retinitis seems to be more generally applicable than any other to the retinal complications of hypertension and nephritis and to permit of more ready comparison of these complications with the other features of the systemic disease.

Wagener and Keith reported in 1923 on a condition which they termed "the malignant hypertension syndrome." The objective findings are persistently elevated blood pressure, diffuseness of arterial and arteriolar thickening throughout the body, minimal changes in parenchyma of kidney and retinal changes. The important retinal alterations are the marked spastic and organic narrowing of the arterioles, with diffuse retinitis and oedema of the discs. The characteristic symptoms are nervousness, asthenia, visual disturbances, dyspnoea on exertion. All observers are agreed that the patients of this group are in very serious condition. A follow-up of 146 cases showed that 80 per cent. of them died within one year.

Dr. Bailliart, dealing with the clinical part of retinal arterial hypertension, stressed the value of his dynamometer which estimates the diastolic blood pressure in the retinal arterioles, which is about 40 per cent. of the diastolic pressure in the brachial arteries. The pressures as measured by this method furnish a ready and probably fairly reliable method of determining variations from the normal in pathological states.

The discussion on endocrinology and its relation to the eye was opened by Dr. Snapper (Holland), Dr. von Szily (Germany), Dr. von J. Imre (Hungary), Drs. Jeandelize and Drouet (France), and Dr. Le Cascio (Italy).

Recent investigations have shown that various disorders of the sexual glands are associated with pathological changes occurring in the eyes. Periodic haemorrhages of the subconjunctival tissue, recurring conjunctival inflammations, or especially phlyctenular kerato-conjunctivitis are not rarely found in close relation to menstruation. Filamentary keratitis following atrophy of the lacrimal glands and often combined with arthritic processes, as a

rule develop in women with ovarian disorders or in the beginning of the climacteric.

Keratoconus has been shown in many instances to be a manifestation of disorders of endocrine glands (thyroid and gonads). Von Imre is of opinion that there is a close connection between acute and chronic glaucoma and abnormal folliculin production. The folliculin production of four glaucomatous patients, examined by reliable biological methods, proved to be exceedingly low. The production of prolan was in every case normal. He points out that there is an undoubted connection between the changes of the intra-ocular tension and the pituitary during pregnancy.

Patients suffering from retinitis pigmentosa are treated nowadays with Menformon and in many cases improvement follows and the progression of the disease is arrested.

At the meeting of the International Organisation against Trachoma Mr. A. F. MacCallan, President of the Organisation, gave an introductory address. Dr. Rowland P. Wilson read a paper on the pathology of Trachoma. The theory of bacterial origin of trachoma has been abandoned. Attention is concentrated mainly on its resemblance to disease caused by known viruses. In Egypt, 90 per cent. of the population is still trachoma infected. The question of reduction of this permanent scourge is closely connected with the social conditions prevailing in Egypt. The excellent work done at present in Egyptian hospitals can only diminish complications resulting from trachoma and thus reduce the incidence of blindness.

At the meeting of the International Association for Prevention of Blindness, Dr. Bailliart (president) read an introductory address. Mr. MacCallan (London) gave an introductory address. Dr. F. Park Lewis (New York) discussed the responsibility of the ophthalmologist in the conservation of sight and the prevention of blindness. The prevention of blindness is as worthy of consideration by ophthalmologists as either research or surgical care in affections of the eyes. The blindness that comes from negligence, from ignorance, from indifference and the crudities of living and its mismanagement is universal. The proportion of eyes that are lost and might be saved by preventive measures is greater and the results better than those in which sight is restored by science and by surgical skill.

Dr. Joseph Minton (London) read a paper on prevention of industrial eye injuries. An investigation was carried out by him at the Royal Eye Hospital, London, where over 7,000 patients are attended to every year suffering from industrial eye injuries. The present high numbers are due to (1) non-provision of safety measures by employers and (2) the attitude of indifference shown by workmen to the risks entailed in their work and their negligence

in using preventive measures supplied to them. Following the above investigation of industrial eye injuries he has organised at the Royal Eye Hospital a museum of preventive measures at the hospital outpatient rooms. The patients have thus an opportunity of studying the preventive appliances at the time of attendance at the hospital and thus choosing the most suitable one for their type of work. If other hospitals situated in industrial centres will take an interest in preventive work, a diminution of eye injuries and with it of its resultant total or partial blindness would follow.

Mr. Lewis H. Carris (U.S.A.) read a paper on the rôle of the social worker in a prevention of blindness campaign. Educators, public health officials, nurses, industrialists, sanitary engineers, social workers and all in any way concerned in human welfare must take their rightful place in this attack upon this social enemy. To the world at large we must teach the causes of blindness and of defective vision, the place of nutrition and hygiene in relation to eye health, methods of safeguarding the eye aginst injury and harm and of keeping it in a state of greatest possible usefulness

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

Executive Committee

A Meeting of the Executive Committee was held on December 9, 1937, at the Semiramis Hotel, Cairo.

The Secretary General read the Minutes of the last Meeting of the Executive Committee held in Paris in 1936. The accounts were examined and found to be correct. The subvention given for purposes of research by the American Academy of Ophthalmology and Oto-Laryngology, 100 dollars, was allocated to Dr. Poleff of the Pasteur Institute at Rabat, Morocco. It was decided to hold the next meeting of the Organization at the same place, and at the same time, as the next Meeting of the International Council of Ophthalmology in the year 1939.

Assembly of Delegates

An Assembly of delegates was held on December 10, 1937, at the Semiramis Hotel, Cairo.

A statement was made by the President as to the business to be transacted. The audited accounts were passed. Professor Manuel Marquez was elected an Honorary Member of the Executive Committee.

The President, Mr. MacCallan, and the Secretary General, Dr. Wibaut, having been elected for a period of five years at the Meeting in London in 1935 remain in office. According to the statutes four members of the executive committee were due to retire in 1935; however, as all members were due to retire by 1937, it was decided to defer elections until the present time in order to obtain the presence of the maximum number of Delegates.

The President explained that six Nations were already represented on the Executive Committee, as follows:—

Great Britain	The President, Mr. MacCallan.	
Holland	The Secretary General, Dr. Wibaut.	
France	Dr. Bailliart	*Ex-officio* as representing International Association for Prevention of Blindness.
U.S.A.	Dr. Park Lewis	
Hungary	Professor de Grosz	Honorary Members.
Spain	Professor Marquez	

There being twelve vacancies on the Committee it was proposed by the President that twelve Nations should be represented other than those previously mentioned, and that each national representative should be selected by each national group. Also that as provided in the Statutes no distinction should be made as to grades of Vice-Presidents, Members and Secretary. This was agreed to.

It was then proposed, seconded and carried that the following Nations, as being the most trachomatous, should be represented on the Executive Committee:—

Argentina; Bulgaria; Czecho-Slovakia; Egypt; Germany; Italy; India; Japan; Lithuania; Poland; Rumania; Turkey.

The Meeting concluded with a vote of thanks to the President.

Subsequently it was announced that the national groups of the above-named countries had selected the following as their representatives:—

Argentina	Dr. Jose A. Sena.
Bulgaria	Prof. Pascheff.
Czecho-Slovakia	Prof. Kadlicky.
Egypt	Dr. Tewfik.
Germany	Prof. Rohrschneider.
Italy	Prof. Leonardi.
India	Dr. Mukerjee.
Japan	Prof. Oguchi.
Lithuania	Dr. Avizonis.
Poland	Dr. Zachert.
Rumania	Prof. Michail.
Turkey	Dr. Naci Bengisu.

The Scientific Meeting

Held at the Semiramis Hotel, Cairo, on December 10, 1937.

(1) Introductory address by the President, Mr. MacCallan.

(2) The microbiological aetiology of trachoma:—

Reporters: Dr. Phillips Thygeson, Prof. Dr. Grüter, Prof. Dr. Oguchi, Drs. Cuénod and Nataf (in their absence an abstract was read by Dr. Farhat), Dr. Rötth, Dr. Julianelle (in his absence an abstract was read by Major Stewart).

Discussion; Major Stewart, Prof. Dr. Igersheimer, Dr. Taborisky, Dr. Postic, Prof. Dr. Favoloro, Dr. Gomez Marquez.

(3) The pathology of trachoma:—

Reporters: Dr. Wilson, Prof. Dr. Michail, Prof. Dr. Mulock-Houwer, Prof. Dr. Pascheff, Dr. Busacca.

Discussion: Prof. Dr. Lauber, Dr. Rötth.

(4) The treatment of trachoma:—

Reporters: Dr. Sobhy Bey, Dr. Shimkin.

Discussion: Prof. Dr. Sabadini, Prof. Dr. Preciosi, Dr. Narog, Dr. Gomez Marquez.

(5) Independent communications:—

Prof. Dr. Motegi: "The epidemiology of trachoma in Japan."

Dr. Lijo-Pavia: "Trachoma among school children in Buenos Aires." (Abstract read by Dr. Bertotto.)

Discussion: Prof. Dr. Barriere, Dr. Sena.

Dr. Zachert: "Application of the ophthalmoscope for the diagnosis of trachoma."

Dr. Sarnelli: "Influence of radiation on trachoma."

Discussion: Prof. Dr. Parparcone.

Dr. Jacovides: "My ideas on trachoma."

Dr. Hassouna: "Treatment of trachoma by tuberculin."

Dr. Charamis: "Plan of a new ophthalmic hospital at Athens."

COUNCIL OF BRITISH OPHTHALMOLOGISTS

Report on Standards of Vision for Candidates for Scholarships and Teacherships under Local Educational Authorities

THE Council appointed the following Committee to review and report on the Standards of Vision required of Candidates for Scholarships and Teacherships under local Educational Authorities which have been in force since 1924:—The President and Hon. Secretary (*ex-officiis*), with Sir Arnold Lawson, Messrs. Percy Flemming, Bishop Harman, A. C. Hudson, P. G. Doyne, W. H. McMullen and Dr. Wiley.

The following report has been agreed upon and is being issued to the Authorities.

In the interests of the community and of the individual it is important that scholarships and teacherships should not be granted to candidates suffering from such ocular defects as may be aggravated by long-continued study and lead to inefficiency or incapacity later in life. It is, therefore, advisable to establish, if practicable, standards of vision which shall exclude unsuitable candidates. At the same time standards should not be fixed so high as to exclude any appreciable number who might in practice prove fit.

The average age of entry upon a junior county scholarship is 11 years; the holder is transferred to a secondary school for five years. Supplementary scholarships are awarded at about 13 years of age, and are held for three or four years at a secondary school. Intermediate scholarships are granted at about sixteen for two or three years. Senior county scholarships are granted at the age of 18 years, and are tenable at a university for three or four years. Prospective teachers undergo training at a college between the ages of 17 and 20 years.

The error of refraction presenting the greatest difficulty in reference to education is myopia.

Myopia is found in about 10 per cent. of children at the leaving age in elementary schools, and in about 10 per cent. of these myopes it becomes a serious defect; in the remaining 90 per cent. of the myopes the defect causes little inconvenience apart from the necessity for wearing glasses. It is generally agreed that in many persons prolonged near work on fine objects under comparatively poor lighting is a cause of myopia, or aggravates existing myopia, especially where astigmatism is present. If preventive measures are to be initiated, and the selection of individuals likely to become unsuitable for such special duties as teaching is to be avoided, it is necessary to determine at as early an age as possible the unfavourable cases. In elementary schools cases of myopia of considerable degree are found in infants under seven years of age, but the first routine test of vision is not made until the children are about eight years old. Of the cases examined at this age, and sent to clinics, some with diminished visual acuity and considerable myopia are transferred to myope classes; others are ordered glasses and return to school, but usually no sufficient effort is made, either at the clinics or at the schools, to warn parents regarding the future of these children.

At the age of eleven the child either is left to continue his education in the elementary school until fourteen years of age, or goes, frequently by means of a scholarship, to a school giving higher education of a more intensive kind. Successful scholarship

candidates are then medically examined, and the first administrative difficulty arises in the elimination of those to be deemed medically unfit.

A child of eleven with 5 dioptres of myopia would by most authorities be deemed unfit for a scholarship. He must continue at the ordinary school, or, exceptionally, go to a myope class. A child of this age with 2 dioptres of myopia would be deemed fit for a scholarship. Between these limits of 5 and 2 dioptres of myopia a decision has to be given on many difficult cases. Many young myopes have good visual acuity with correcting glasses, and apparently healthy fundi, yet their defect is often progressive. The determination in all young myopes of the rate of progression is most important. The previous history, however, is often defective. It may be known that the child has worn concave glasses for some time, or, at best, there may be a record that vision was tested at the age of eight and found normal. More accurate records would be of great value, and it is desirable that such should be kept.

Astigmatism of considerable amount may cause want of efficiency, especially in those engaged in an occupation such as teaching, which involves much close or fine work. With very high degrees of this defect, visual acuity, even with correcting glasses, is usually subnormal.

The recommendations of the Council are as follow:—

Visual acuity.—In all candidates for scholarships and teacherships visual acuity, with correcting glasses, should not be less than 6/9 in the better eye.

Myopia.—A child of eleven with not more than 4 dioptres of myopia in the better eye should be passed for scholarship training; and children with a higher degree than 4 dioptres should be referred to an ophthalmic surgeon for an opinion as to fitness.

At the age of fifteen those with not more than 5 dioptres in the better eye should be deemed fit to train for the teaching profession. Those with higher degrees than 5 dioptres should be referred to an ophthalmic surgeon for an opinion as to fitness.

Candidates for entry to training colleges, who are as a rule about eighteen years of age, if they have not more than 6 dioptres of myopia in the better eye should be accepted. Those with higher degrees than 6 dioptres should be referred to an ophthalmic surgeon for an opinion as to fitness.

Special attention should be given to the physique and general health of all myopic candidates. A border line case associated with poor physique should be rejected as the risks of increase in myopia are greater in such cases.

Special cases.—If there is only one eye, or if there is only one useful eye, the other being amblyopic from conditions which in no

way affect, nor are likely to affect the good eye, the case should be judged on the condition of the good eye.

In the case of candidates for university and technical senior scholarships, or those intending to specialise as teachers of certain technical subjects, greater latitude may be allowed after consideration of special circumstances as to nature of work, condition of the candidate's eyes in other respects, etc.

BOOK NOTICES

The Transactions of the Ophthalmological Society of the United Kingdom. Vol. LVII. Pt. 1. 1937.

Volume LVII of the *Transactions of the Ophthalmological Society of the United Kingdom* has been divided into two bound parts. Part I contains a list of the Society's Officers and Council, Honorary Members, the general list of members, the rules and the papers read at the Annual Congress, and the annual lectures and papers read before the North of England Ophthalmological Society.

In this part there is also the valuable and interesting Presidential address which was given by Dr. Gordon Holmes on the prognosis in papilloedema.

Among the scientific contributions those of Professor Woollard, Dr. Lythgoe and Dr. Goodeve deserve special mention.

The discussion on the rare forms of keratitis is of interest, so are Professor Ballantyne's lectures on modern methods in ophthalmoscopy and the evolution of retinal vascular disease.

We hope to publish abstracts of some of these papers during 1938.

Part II will contain the papers read before the Oxford Ophthalmological Congress (including the Doyne Memorial Lecture) and the transactions of the other Affiliated Ophthalmological Societies.

Post-Graduate Surgery. Edited by RODNEY MAINGOT. Vol. III. Pp. 3575-5584, Figures 1981-2994. London: Medical Publications, Limited. 1937.

Volume III of Post-Graduate Surgery contains Parts XVII to XXXIX, the majority of which are concerned with special departments such as obstetric surgery, ophthalmology, orthopaedics, otorhinolaryngology, plastic surgery, odontology, venereal diseases and others. There is also an interesting account of the medical aspects of surgery, physical medicine and deep X-ray therapy. The whole work is admirably produced and the illustrations are especially good. It it almost inevitable in the compilation of such a work that some lack of balance should occur.

The section on diseases of the eye and ophthalmic operations has been ably contributed by Williamson-Noble who has compressed this subject into 77 pages. In a lucid and concise manner he has given an account of the methods of examination and the salient features in certain common ocular disorders. There are many admirable illustrations of ophthalmic operations but a few of these are open to criticism on account of artist's errors. In Fig. 2451 showing Greeves's operation for ptosis the conjunctival incision is much too short and if anything a little low. In the text describing this operation no caution is given concerning the avoidance of damage to the insertion of the levator palpebrae superioris in cutting the tongues from the upper margins of the tarsal plate. The stitches are not removed as the text states but are left *in situ*. There is also no mention of a troublesome complication after this operation, inversion of the upper lid margin due to kinking of the strips from the tarsal plate.

Fig. 2462 shows a glaucoma iridectomy and the section is that used for cataract extraction, and too large for this operation. Fig. 2450 illustrates the Kuhnt-Szymanowski operation at a late stage. One or two diagrams of the early phases of the operation would make it easier for the student to understand.

In the description of the dacryocystectomy operation the author advises an inspection of the cornea at the end of the operation for abrasions and scratches, but he has omitted to state that this undesirable complication could be avoided by temporary suturing of the lids before the operation is begun.

The print is good and the work fulfils its purpose in giving post-graduates an introduction to the commoner disorders of the eye and ophthalmic operations.

Routine Methods of Treatment as employed from time to time in the Government Ophthalmic Hospital, Madras. By Lieut.-Col. R. E. WRIGHT, Superintendent, Government Ophthalmic Hospital, and RAO BAHADUR K. KOMAN NAYAR, Assistant Superintendent, Government Ophthalmic Hospital, Madras.

This small manual sets out in a convenient and lucid manner notes on the routine treatment of patients admitted to the Government Ophthalmic Hospital, Madras.

It is fairly wide in its scope and deals with the care of malaria, leprosy, the doses and modes of administering diphtheria antitoxin and anti-tetanic serum. Several pages are devoted to the non-operative treatment of primary glaucoma including a variety of drugs and intravenous hypertonic salines.

Considerable attention has also been paid to the treatment of syphilis, full details are given of the courses of treatment.

The use of acetyl choline and nitroscleran in the treatment of

arterial spasm and ischaemia is described briefly. The treatment of intestinal worms is given quite fully.

Generalised narcosis is of topical interest and this book sets out the details of induction of anaesthesia by rectal paraldehyde, sodium evipan, avertin and rectal ether.

There is much that will interest the ophthalmic surgeon in this small manual.

The pharmacopoeia contains a list of ophthalmic and general medical preparations with some brief foot notes about dispensing and the sterilisation of drops. Blank pages are left interspersed throughout the booklet for the purpose of making notes.

Annual Report and Statistics of the Government Ophthalmic Hospital, Madras, for the year 1936.

The report contains a list of the official staff of the hospital and an account of its accommodation and financial state.

31,609 out-patients received treatment and 4,391 were admitted to the in-patient department during 1936. There is also a list of the diseases treated among both in-patients and out-patients, and statistics of medical students and post-graduates trained at the hospital.

The list of operations performed during the year is especially interesting. Cataract extraction exceeds all others, the intra-capsular extractions number 336 and extra-capsular 1,938. Only 4 operations were done for strabismus and 14 for retinal detachment.

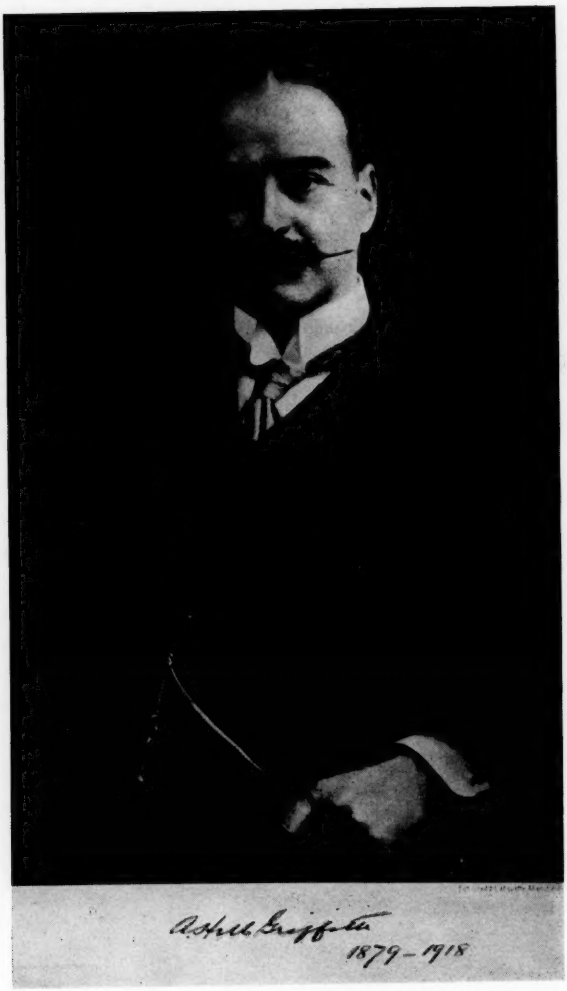
CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—I would like to hear readers' opinions of this criticism of the term "Ophthalmic Surgeon."

The word "Ophthalmic" is difficult and conveys little, even to people of average intelligence. Anyone can verify this on a number of non-medical people of average education. Those of the public who appreciate that "Ophthalmic" has to do with eyes, are likely to confuse Ophthalmic Surgeon with Ophthalmic Opticians, who appear to have "borrowed" the title, no doubt to be in good company. As the genus that includes Ophthalmic Opticians, Optometrists, Eye Specialists and all such, is widely distributed and advertised, confusion is inevitable.

Why not therefore, discard the term "Ophthalmic" Surgeon and substitute "Eye" Surgeon—("Surgeon Oculist" is a possible



From an enlargement in the Royal Eye Hospital, Manchester

UNIV. OF MICH.

alternative), "Eye Surgeon" is self-explanatory and further, it suggests the use of surgery in the treatment of eye diseases. Compare the simplicity of "Brain Surgeon," with say, a title like "Cephalic Surgeon." The term "Eye Surgeon" would be difficult to simulate or "borrow."

I am,

Yours truly,

C. CONOR O'MALLEY.

6, THE CRESCENT,
GALWAY.
December 15, 1937.

OBITUARY

A. HILL GRIFFITH

WE regret to announce the death of Dr. Alexander Hill Griffith, which occurred at Woking on November 25, 1937, in his seventy-ninth year.

He was born in Aberdeen and was the seventh son of Charles Fox Griffith, J.P. He was educated at the Aberdeen Grammar School and University of Aberdeen and passed his final examinations for the degree of M.B., C.M., before he was twenty-one years of age. After his qualifying examination (April, 1879) and before he received his M.B. degree in April, 1880, he was appointed House Surgeon at the Manchester Royal Eye Hospital, a position which he held for nearly seven years. At that time little was done for the treatment of astigmatism and it was principally due to Griffith that adequate attention was given to this branch of refraction. Again during the time of his residence he was very interested in fields of vision and as a result of his investigations he wrote an excellent paper on this subject in the *Manchester Medical Chronicle.*

In 1883 he obtained the degree of M.D. In 1886 Griffith was appointed to the Honorary Surgical Staff of the hospital, first as assistant surgeon and three years later to the full staff. He was now in a position to pursue his clinical investigations and perfect his technique. This was supplemented by his love for histology. He was a keen microscopist and cut all his own sections. In 1896 he took the F.R.C.S.(Edin.). His contributions to our literature evidently attracted the attention of his contemporaries, for in 1897 he was awarded the Middlemore prize by the British Medical Association.

In 1898 he wrote the section on diseases of the choroid and vitreous in Norris and Oliver's text book of ophthalmology. This was an excellent contribution on these subjects and amongst many interesting points he expressed views on the permeability of the suspensory ligament of the lens, a subject noted by him in a previous communication to the Ophthalmological Society.

In his chapter on choroidal disease he emphasized the importance of transillumination as an aid to the diagnosis of choroidal neoplasms.

In 1899 Griffith was appointed Honorary Ophthalmic Surgeon to The Manchester Royal Infirmary and later lecturer on ophthalmology to the University of Manchester.

He joined the Ophthalmological Society in 1885, was on its committee from 1893 to 1896 and later was a Vice-President (1905 to 1908). For many years he was a member of the Manchester Medical Society and made several valuable contributions to it. For forty-three years he was a member of the British Medical Association was its secretary to the section of Ophthalmology at the annual meeting held in Manchester in 1902, and was Vice-President at the Belfast Meeting in 1909. He was one of the founders of the North of England Ophthalmological Society and was its first President.

On his appointment to the staff of the Manchester Royal Infirmary he was brought into contact with the students and it was soon apparent that his abilities as a teacher were great. His enthusiasm was immense and the way in which he investigated a case together with his sound reasoning were greatly appreciated. To one who was a student under him and subsequently his House Surgeon and colleague it is difficult to write about Griffith's work without the fear of exaggeration. Mention has been made of his teaching ability but when one was in a better position to appreciate his qualities as a clinician it became apparent that he was a master of his craft. He was a careful observer, his deductions were sound and convincing and he was a wizard with the ophthalmoscope. Griffith was an excellent operator. It was really delightful to watch his technique in such an operation as iridectomy for glaucoma for no anterior chamber seemed too shallow for him to enter with a broad keratome. Again, his cataract operations were beautifully executed and his originality is well illustrated by the novel manner in which he approached the corneo-sclera when trephining for glaucoma—a technique mentioned in Elliot's book on this subject.

As a colleague he was delightful to work with and he was always ready to help with his advice especially in difficult cases. Not only was he very much appreciated by his students and colleagues but was looked upon by his patients as a kind and helpful friend.

In addition to the large amount of hospital work he had a very extensive practice. He held the rank of Major R.A.M.C.(T.) and

during the whole of the Great War gave valuable aid to the large number of wounded soldiers under treatment in Manchester.

He retired from practice in 1918 and went to live in Woking where he proceeded to enjoy his three great hobbies, singing, billiards and golf. He had always been attached to music and on his retirement he took singing lessons at regular intervals and proceeded to take part in musical competitions. How great was his delight when he showed one the marks he had gained in a singing competition and he took considerable interest in the criticisms of the adjudicators. He enjoyed a game of golf and at one time he was captain of the Manchester Golf Club (medical).

Early last year his health began to fail and a motor accident in the early summer may have caused further weakness. But there was still energy left, for he insisted on going up to London for a singing lesson a fortnight before he died. On his return from London he was so weak that he entered a nursing home and died as stated on November 25.

He was cremated at Woking and his ashes were scattered in St. Mary's Churchyard, Horsell, near Woking.

NOTES

Honour

THE Royal Society of Medicine of Budapest, on the occasion of the centenary of foundation, has elected Sir John Parsons and Mr. Leslie Paton honorary members.

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The Ophthalmological Society of the United Kingdom

THE Annual Congress will be held in London on April 28, 29, 30, 1938. The subject for discussion is "The differential diagnosis of the causes of exophthalmos." Openers will be Mr. Foster Moore (Ophthalmology), Mr. T. E. Cawthorne (Ear, Nose and Throat), Dr. Russell-Brain (Medicine and Neurology).

Members who wish to take part in the discussion or to read papers should communicate with Mr. L. H. Savin, 7, Queen Street, Mayfair, W.1, if possible before February 28, 1938. Abstracts of papers, for circulation before the meeting, should be sent before March 27.

A visit will be arranged to White Oak Hospital, Swanley, by Mr. Arnold Sorsby, if a sufficient number of members send in their names before February 28. On the afternoon of April 30, the Post Office authorities have invited members of the Congress to visit their headquarters where the sorting offices, the Central Telegraph

Office and the underground P.O. railway will be shown. Tea will be provided. The Annual Dinner will be held on Thursday, April 28.

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Canadian Ophthalmological Society

AT a recent meeting held in Toronto the Canadian Ophthalmological Society adopted a Constitution and Bylaws. Dr. W. Gordon M. Byers' untiring work in connection with the formation of the Society has been rewarded by a membership of sixty of the leading practitioners doing eye work and representing every Province in the Dominion.

The following officers were elected:—President, Dr. W. Gordon M. Byers; Vice-President, Dr. W. H. Lowry; Secretary-Treasurer, Dr. Alexander E. MacDonald; Members of the Council, Dr. F. T. Tooke, Dr. Colin Campbell, Dr. J. Vaillancourt, Dr. E. A. McCusker, Dr. L. de V. Chipman.

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Royal London Ophthalmic Hospital

THE Annual Dinner of Past and Present Students of the Royal London Ophthalmic Hospital, will be held at the Langham Hotel, Portland Place, on Thursday, March 10, 1938, at 7 for 7.30 p.m. Chairman: A. C. Hudson, Esq., M.D., F.R.C.S., Consulting Surgeon to the Hospital. Tickets 15/- (excluding wine). An early reply is requested to: Mr. P. G. Doyne, 60, Queen Anne Street, W.1. Great inconvenience will be avoided if remittance accompanies application. Secretaries: Charles Goulden, 89, Harley Street, W.1, and P. G. Doyne, 60, Queen Anne Street, W.1.

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International Council of Ophthalmology

AT the meeting on December 14, 1937, Mr. A. H. H. Sinclair, of Edinburgh, was elected a member of the Council in place of Sir John Parsons and Mr. A. F. MacCallan was elected a member—*ex-officio*—as President of the International Organization against Trachoma.

FUTURE ARRANGEMENTS

1938

February 1.—Midland Ophthalmological Society, at Wolverhampton Eye Infirmary.

February 4.—North of England Ophthalmological Society, at Newcastle-upon-Tyne.

February 11.—Section of Ophthalmology, Royal Society of Medicine.

March 4.—North of England Ophthalmological Society, at Liverpool.

March 11.—Section of Ophthalmology, Royal Society of Medicine (Clinical Meeting).

April 1.—North of England Ophthalmological Society, at Sheffield.

April 8.—Midland Ophthalmological Society, at Nottingham Eye Infirmary.

May 27.—Midland Ophthalmological Society, at Cheltenham Eye Hospital.

June 10.—Section of Ophthalmology, Royal Society of Medicine (Annual Meeting).

July 7-9.—Oxford Ophthalmological Congress, at Oxford.

October 7.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *November. 1937.*

WRIGHT. Fatty degeneration of the cornea (neutral and lipoid).

CARMODY. Herpes zoster ophthalmicus complicated by ophthalmoplegia and exophthalmos.

LEHRFELD and REBER. Glaucoma at the Wills Hospital, 1926-1935.

COGAN. Accommodation and the autonomic nervous system.

GOLDSTEIN and WEXLER. Bilateral atrophy of the optic nerve in periarteritis nodosa: a microscopic study.

GOUGELMAN. Fitting of protheses for patients with cryptophthalmos and extreme microphthalmos.

CLAY and BAIRD. An unclassified type of optic neuritis: report of cases.

FERREE and RAND. Human factor in airplane crashes.

HAESSLER. Near reaction of the pupil in the dark: a quantitative study.

CURDY. Equal advancement and recession operation for horizontal strabismus.

KRAUSE. Chemistry of the retina. IV. The bacillary layer.

LEVITT, Chronic oedema of the cornea: report of a case.

FELDMAN. An instrument for qualitative study of dark adaptation.

December, 1937.

WAGENER and CUSICK. Chiasmal syndromes produced by lesions in the posterior fossa.

WHITE. Muscular anomalies and ametropia: a practical consideration.

DUTT. Lever action operation for intra-capsular extraction of cataract.

BORLEY and TAINTER. Effects of dinitrophenol on the permeability of the capsule of the lens.

WALLS. Significance of the foveal depression.

APPELBAUM. Chancre of the upper eyelid in an infant two months of age: report of a case.

DIMITRY. Use of iodized poppy seed oil in ophthalmology.

ZIPORKES. A case of mixed tumour of the lacrymal gland, with retinal folds and choroidal detachment, which disappeared after the removal of the growth.

ALLAN. Eugenic significance of retinitis pigmentosa.

CARROLL. Importance of diet in the aetiology and treatment of tobacco-alcohol amblyopia.
CALHOUN. Intra-ocular invasion by the larva of the ascaris; report of a case with unusual complications.
DVORAK-THEOBALD. Neurogenic origin of choroidal sarcoma.

American Journal of Ophthalmology. *December, 1937.*

SMELSER. Experimental and clinical exophthalmos.
MCFARLAND, KNEHR and BERENS. Effects of anoxemia on ocular movements.
LANCASTER. Lighting standards.
BORLEY and LEEF. Removal of lead shot from the vitreous.
BARKAN, O. The surgery of chronic glaucoma.
MONCRIEFF. Cataract surgery.
HILDRETH. Method of preparing a transparent eyeball.
BROWN. Vision after glaucoma operation.

Annales d'Oculistique. *November, 1937.*

HAMBRESIN. Pyretotherapy in ophthalmology.
MIRANDA. Myelinated retinal fibres.

Revue d'Oto-Neuro-Ophtalmologie. *November, 1937.*

TILLÉ. Glaucoma—labyrinthine syndrome. Some observations of vertigo, deafness and buzzing associated with glaucoma.
ROGER and FARNARIER. The Laurence-Moon-Bardet-Biedl syndrome. A personal case.
JEAN-SÉDAN. Recurrent transient hemianopsia, of a menstrual character from cortical and retinal angiospasm with syphilitic hypertensive arteritis.

Revue Internationale du Trachome. *October, 1937.*

ADAMENTIADIS. Several clinical questions concerning trachoma.
MASSOUD. Trachoma and prophylaxis for children.
CORNET. Trachomatous and non-trachomatous elements in patients affected with trachoma. Morbid associations.
ROSTKOWSKI. Does follicular conjunctivitis present a particular morbidity?
CORNET. Syphilitic palpebro-conjunctivo-corneal elements in the trachomatous.
DELANÖE. Experimental and clinical research on the propagation of trachoma.
TRAPESONTZEWA. Trachoma as an infection.
SEGAL-CARITAS. Therapeutic note on Cusylol.

Archiv für Ophthalmologie. *December, 1937.*

GREEFF. What knowledge do we possess of von Graefe? Three unknown representations of von Graefe.
MIKLOS. Treatment of tabetic optic atrophy.
DERKAC. Trial of a specific treatment for trachoma.
OHM. Nystagmus.

Rassegna Italiana d'Ottalmologia. *July-August, 1937.*

BESSO. A case of acute bilateral oedema of the orbit with resultant atrophy of the optic nerve following administration of potassium iodide.
NASTRI. Contribution to the study of the genesis of idiopathic retinal detachment.
RUSSO. Primary adiposity of the cornea.
BOSSALINO. Further contribution to the knowledge of tumours of the ciliary body and of the iris.
AGNELLO. Reticulo-plasmocytoma of the conjunctiva and its association with degenerative trachoma.
FARINA. Histopathogenesis of retinitis pigmentosa.